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A Renal Dialysis and Kidney Transplantation Program for the State of Maine

A Progress Report

DONALD F. MARSHALL, M.D., JAN DREWRY, M.D. and FERRIS S. RAY, M.D.

Several years ago a national survey was carried out showing that a dialysis and transplantation center should be made available for every one million population to care for patients suffering from end-stage kidney disease.

After considerable deliberation and with the cooperation of the Honorable Kenneth Curtis, Governor of Maine, the Maine State Legislature, numerous dedicated persons, the Board of Directors and Hospital Administrators of the Maine Medical Center collaborated in the establishment of a dialysis and kidney transplantation center at the Maine Medical Center. The State Legislature allocated $500,000 for these procurement authorities at the Maine Medical Center to set apart ten rooms in the new Richards Wing for renal dialysis and transplantation.

The Dialysis Center is under the direction of Dr. Jan Drewry and is staffed by six nurses and three laboratory technicians. As of July 1, 1972, a second Nephrologist, Dr. Donald Leeber was added to the staff. Through the generosity of Mr. Albert Conley of Freeport, Maine, a Tissue-Typing Laboratory is currently being equipped and staffed by trained technicians under the direction of Dr. Drewry.

Four transplant teams made up of four general surgeons and four urologic surgeons under the direction of Dr. Ferris Ray have completed three successful kidney transplantations from living related donors and one cadaveric transplant to date.

Sixteen kidneys have been saved from victims of fatal auto accidents, preserved and transported to other areas of New England for successful transplantation.

It should be emphasized that this center in Portland has been established to benefit all sections of Maine and for all Maine people suffering from end-stage kidney disease.

The treatment of patients with chronic renal failure has evolved over the past five years from two separate programs, i.e., dialysis or transplantation, toward a more unified approach to the long-term care of these patients. With the accumulation of more experience in the fields of dialysis and transplantation, several facts have become apparent:

(1) Transplantation is a feasible mode of therapy for larger numbers of patients than originally anticipated.

(2) Long-term chronic dialysis is also feasible due to its general accessability and relative decrease in cost over recent years.

(3) With a combined program of dialysis and transplantation, interchangeable modes of therapy are available to most patients with end-stage renal disease.

The approach to the therapy of a patient involves, by necessity, long-term planning. The initial evaluation of such a patient consists of not only medical evaluation, but psychological, social and financial evaluations. The treatment can be then structured so as to best suit the individual patient's needs.

In-hospital dialysis, as it exists not only in the State of Maine but throughout the entire country, has been shown to be acceptable only under certain circumstances. These circumstances include holding for transplantation, preparation for the home training program, back up dialysis for home patients who become ill, and the treatment of patients with acute renal failure.

The dialysis unit at Maine Medical Center utilizes 9 Travenol RSP dialysis machines. With the use of this system, patients are dialyzed three times weekly for 6 hours. This permits the potential of dialyzing 27 patients

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each day if the unit were operational for 24 hours.

Some patients can be trained to dialyze themselves at home with the help of an assistant. This arrangement has the advantage of minimum disruption of normal life patterns and lower cost. The training period lasts in general eight weeks and is carried out under the direction of Dr. Drewry within the hospital dialysis unit. During this period, patients gradually assume the responsibility for dialyzing themselves. Twenty-four hour a day back up assistance by physician, nurse, and technician is provided for these patients should the need arise after they have returned home. Home dialysis, in addition to being the only appropriate treatment for some patients, also serves a holding function for those patients awaiting cadaveric transplantation.

As of July 1, 1971, one year after the opening of the dialysis unit, seven patients were being dialyzed. Of those seven patients, four have subsequently been transplanted, one was subsequently trained for home dialysis, and one remains in the unit awaiting cadaveric transplantation. In the period from July 1, 1971, to July 1, 1972, eleven new patients have been admitted to the chronic dialysis program. Of these eleven, three have received kidney transplantation and five have been home trained. Four patients remain within the unit. Two await cadaveric transplantation, and two related living donor transplantation. During this same period of time, twenty-one patients with acute renal failure have been treated with hemodialysis.

In the four-month period from July 1, 1972, to the preparation of this report, ten new patients have already been admitted to the chronic dialysis unit. Of these, three are completely home trained and dialyzing at home, while four are currently in the home traning program. In the same four-month period, ten patients with acute renal failure have been treated with dialysis.

At the present time, a new five-bed home dialysis training center is under construction in conjunction with a tissue typing laboratory to facilitate cadaveric and related living donor transplantation in these patients.

The present concept concerning the incidence of chronic renal failure is that forty new patients each year per million population will require some form of therapy for end-stage kidney disease. Under the present system and projected plans, the dialysis unit will be able to meet this growing need and provide acute and chronic dialysis, home training, in hospital "backup" facilities, and a transplant holding facility for these patients.

Although chronic hemodialysis has dramatically prolonged the survival of patients with terminal renal failure, it imposes rigid restrictions upon their lives in addition to an economic burden.

During the past decade, renal transplantation has rapidly become the acceptable therapy for selected patients with terminal renal failure. In spite of many problems yet to be solved, transplantation offers some patients the very best chance for normal health and complete rehabilitation.

Results of Renal Transplantation

Since the development and clinical use of the immunosuppressive drug, Azathioprine (Imuran®) in 1963, the results of renal transplant surgery have improved steadily. Until that time only scattered successes were reported except in cases of identical twins. Azathioprine and Prednisone are the mainstays in immuno-suppressive therapy used routinely in all cases. Although many factors would appear to influence the results, the overall success rates today for kidney survival following transplantation are somewhat as follows.

Donor	*One Year*	*Two Years*	*Three Years*
Living related	90%	85%	80%
Cadaver	85%	75%	50%

The long-term results in cadaver kidney transplants are not nearly as good as those in living related donors, although more recent experience with the use of antilymphocytic globulin (ALG) in these cases appears very promising. There is considerable activity and progress in most large transplant centers investigating the results of ALG and similar preparations. Further refinements and standardization of these drugs together with improved techniques of tissue-typing will no doubt greatly increase the success rate in the cadaver group.

New England Regional Kidney Program

Unfortunately, only about one out of three recipients awaiting renal transplantation will have an acceptable related living donor. For this reason an increasing number of recipients are waiting long periods of time for a suitable cadaver kidney to become available.

In an effort to obtain as many cadaver kidneys as possible for transplantation, a New England Regional Kidney Program was organized in 1970. Working closely with the Inter-Hospital Organ Bank in Boston, a regional program for organ procurement and distribution has been established.

Patients waiting for cadaver kidneys are typed and registered with the Organ Bank in Boston. When a suitable kidney becomes available, the recipient that best matches the kidney is notified and the kidney shipped to the appropriate transplant center. Kidneys have now been exchanged throughout all parts of the New England area and successfully transplanted. It has been shown that with ordinary cold perfusion techniques, a cadaver kidney will function satisfactorily eight or ten hours after being removed. When longer periods of time are necessary between removal of the kidney and transplantation, perfusion machines have proved to be a most satisfactory method of preserving the kidney. Such a machine will be functioning at the Maine Medical Center within the next few weeks. With such equipment, the transplant team will be able to adequately preserve kidneys for up to 36-48 hours.

Continued on Page 4

Cervical Thymic Cyst

A Consideration in the Differential Diagnosis of Neck Masses

KATHLEEN A. SHEPARD* and J. DANIEL MILLER, M.D.**

The differential diagnosis of unilateral neck masses is frequently difficult, particularly in the pediatric age group. Although the possible etiologies usually narrow down to either infection or neoplasm, embryologic remnants must also be considered. The following case of a cervical thymic cyst is presented to emphasize that thymic remnants must be considered in the differential diagnoses of neck masses.

CASE PRESENTATION

G. W. (Hosp. #108725), a 14-year-old white male presented in the Maine Medical Center Pediatric Clinic on March 23, 1972 with a chief complaint of a lump in the left anterior neck. He was active and alert with no generalized lethargy and reported no recent history of sore throat, upper respiratory or oral infection. There was a past medical history of a questionably positive tine test one year prior to the present illness. However, there was no family history of tuberculosis and a follow-up PPD was negative. Review of systems was unremarkable and there was no history of dysphagia, weight loss, fever, chills, night sweats, chronic cough or shortness of breath.

Physical examination revealed a well-nourished, well-developed adolescent male in no distress. There was an oval mass in the left anterior cervical region which was 1.5 cm. x 1 cm. in size, firm, non-tender and fluctuant. There were no palpable nodes in other regions of the body and the remainder of the physical exam was completely normal. The clinical impression was that of an enlarged lymph node. Complete blood count, differential count, sedimentation rate, mono spot test, SMA 1260 (Ca++, BUN, inorg. phos., glucose, SGOT, LDH, alk. phos., albumin, total protein, cholesterol, uric acid and total bilirubin), chest x-ray and PPD skin test were all normal. A course of watchful waiting was decided upon. On return one month later, the mass was no longer palpable; but approximately three months from the first time seen, the patient again presented in the clinic with a left anterior cervical mass, 1.5 cm. x 2 cm., again firm, non-tender and fluctuant. Shoddy nodes were palpable in the axillary and inguinal regions but were not felt to be pathological. The patient was still completely asymptomatic. Repeat complete blood count, differential count, sedimentation rate, leukocyte alkaline phosphatase, T 4, mono spot test, chest x-ray, and PPD (intermediate strength) were all negative. On return visit one week later, the mass was still palpable and the size unchanged but the chest x-ray showed mild hilar adenopathy. The patient was scheduled for surgical evaluation and possible excisional biopsy. The appointment was missed but the patient reappeared two weeks later, at which time the mass was noted to have decreased in size to 1 cm. x 1 cm. Repeat chest x-ray was normal. He was seen in Surgical Clinic five days later and an excisional biopsy was performed under local anesthesia.

PATHOLOGY REPORT

Gross examination revealed a soft, irregular nodule, measuring 2.2 cm. in greatest dimension, and containing multiple discrete and confluent yellow-grey foci on section. Microscopic examination of sections demonstrated a multiloculated cystic structure. The cysts were lined by flattened or squamous epithelium, and were filled with a mixture of desquamated cells, foamy macrophages and foreign body giant cells centered around circular clefts. They were supported by moderate amounts of fibrous stroma, communicating with subcutaneous tissue which formed a wall partially composed of multiple small remnants of thymic tissue containing Hassall's corpuscles. The pathologist's impression was: Cervical Thymic Remnants with multiple cyst formation.

DISCUSSION

In the differential diagnosis of a cervical mass in pediatric patients, infections and neoplasms are paramount. Among the congenital lesions, thyroglossal duct cysts (midline) and branchial cleft cysts (lateral) are most commonly found, with thymic cysts receiving little, if any, mention.

Embryologically, thymic primordia appear late in the sixth week of development as evaginations of the third pharyngeal pouch. They then undergo an elongation process, while still attached to the pharyngeal pouches, and during the seventh week lose their lumina, continue to enlarge and fuse in the midline caudal to the thyroid primordia. This fused tissue mass then gradually descends under the sternum into the anterior superior mediastinum.[1] During the course of descent from the angle of the jaw to the suprasternal notch, it is possible for a nodule of thymic tissue to separate from the main gland, therefore remaining in the path of descent where it may undergo cystic change and present as a cervical mass. These masses are classically benign uniloculated or more often multiloculated cysts found to contain brown, clear fluid with multiple yellow granules. They are often lined with flattened, squamous, cuboidal or cylindrical cells. Thymic tissue with identifiable Hassall's corpuscles is usually found in the wall of the cyst.[2]

Loupalt reported the first case of a congenital thymic cyst, found on autopsy of an 18-year-old female in 1897.[3] The first successful complete excision of a cervical thymic cyst was reported by Hyde, Sellers and Owen in 1944.[4] In a review of literature and case report by Barrick and O'Kell in 1969, 38 cases of thymic cysts were recorded; of these, 30 were located in the cervical region, the remaining 8 in the mediastinum. Of the 28 cases reported in the pediatric age group, the majority were cervical, with only 1 being located in the mediastinum. The majority of cervical thymic cysts are unilateral and predominantly left-sided, but bilateral masses have also been recorded.[5,6] The relatively large number of thymic cysts found in childhood is probably due to the fact that the thymus has its greatest growth between the ages of 2 and

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4 and does not reach its maximum size until puberty.

In the majority of reported cases, patients have been asymptomatic. Tracheal deviation, respiratory obstruction requiring tracheotomy, dysphagia and infection have all been reported as a result of thymic cysts.[6] Malignancy was noted in three cases.[6,7,8,9] Noteworthy in the case reported by the authors was the intermittent character of the palpable mass, a finding which was inconsistent with most of the diagnoses considered. No other reported cases described this striking finding.

In light of the literature reviewed and the case reported, the authors offer the following recommendations: (1.) Thymic remnants should be considered in the differential diagnosis of a cervical mass which persists without signs and symptoms of infectious etiology. (2.) The physical finding of a neck mass which seems to wax and wane may be a clue to the diagnosis of a cervical thymic cyst. (3.) Excisional biopsy should be performed, especially in light of the potential malignancy of cervical thymic cysts.

Summary

A case presentation of a cervical thymic cyst with intermittent character has been reported along with a brief review of the embryology and literature on the subject in an attempt to re-emphasize the importance of considering this entity in the differential diagnosis of an anterior cervical mass, especially in the pediatric age group. The importance of making the diagnosis and of surgical removal is reiterated by the fact that these masses may have potential for malignancy.

Acknowledgment

We wish to express our appreciation to Dr. George W. Hallett, Jr. for reviewing the manuscript and to Dr. Joseph Fanning for assistance in evaluation of the pathological specimens.

References

1. Patten, B. M.: Human Embryology, 3rd ed. McGraw-Hill Book Co., N.Y. 1968, pp. 438-439.
2. Nelson, W., Vaughn, V. and McKay, R. J.: Textbook of Pediatrics, 9th ed. W. B. Saunders Co., Phil., 1969, pp. 1442-1443.
3. Podolsky, S., Ehelick, E. W. and Howard, J. M.: Congenital thymic cyst attached to the pericardium: Case report and case review of literature. Dis. Chest., 42: 642-644, 1962.
4. Hyde, T. L., Sellers, E. D. and Owen, M.: Thymic Cyst of the neck. Texas Med. 39: 539-540, 1944.
5. Barrick, B. and O'Kell, R. T.: Thymic cysts and Remnant Cervical Thymus., J. Ped. Surg. 4: 355-358, 1969.
6. Lewis, M. R.: Persistence of the thymus in the cervical area. J. Ped., 61: 887-893, 1962.
7. Behring, C. and Bergman, F.: Thymic cyst of the neck, Acta Path. Microbiol. Scand. 59: 45-50, 1963.
8. Fielding, J. F. et al: Cystic degeneration in persistent cervical thymus., Canad, J. Surg. 6: 178-186, 1963.
9. Ozlo, C. and Allen, J. D.: Unilateral Thymic cyst of the neck., J. Kentucky Med. Assoc. 60: 743-745, 1962.

A RENAL DIALYSIS AND KIDNEY TRANSPLANTATION PROGRAM FOR THE STATE OF MAINE: A PROGRESS REPORT — *Continued from Page 2*

Kidney Procurement Program in Maine

During the past year, considerable effort has been made in Maine and parts of New Hampshire to inform the public and medical profession of the need of cadaver kidney donation. Meetings with medical staffs in Lewiston, Waterville, and Bangor have been held and interested doctors in these areas and others have been informed of the proper procedure to follow when a potential kidney donor becomes available.

To insure a high degree of success in cadaver kidney transplants, the kidney must be removed as soon as possible after death of the patient. For this reason, early identification of patients is desirable. The most common kidney donor will be patients with irreversible intracranial injury. Although occasional exceptions may be made, acceptable donors must fulfill the following criteria.

1. Age usually under 65.
2. No diabetes.
3. No significant previous hypertension.
4. No bacteremia or septicemia.
5. No malignancy (except primary intracranial malignancy).
6. No hepatitis or active TBC.
7. No chronic renal disease or infection.
8. Acceptable BUN, creatinine, and urinary output.

When a potential kidney donor is identified, the Nephrology Department at the Maine Medical Center (telephone: (207) 871-2417) should be called and the patient discussed with the doctor on duty. A Kidney Procurement Team has been organized and is available to go to the community hospitals to aid in kidney procurement.

There still remains considerable organization and work to be done. Further meetings and closer communications with the staffs and administration of community hospitals is needed.

We hope that through their cooperation and help an increasing number of cadaver kidneys will become available and many patients now waiting long periods of time will receive the benefits of transplantation.

By necessity, both the dialysis and transplantation program require the cooperative effort of many people. It is hoped that with the participation of interested physicians throughout the State, the program for treatment of end-stage renal disease in the State of Maine will continue to grow.

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Survival of a Diabetic Patient With Perirenal Emphysema

Ulrich Ehrig, M.D., F.R.C.P.(C.) and William T. W. Clarke, M.D., F.R.C.P.(C.)

Abstract

A diabetic woman with an ileal conduit developed pyelonephritis and perirenal gas collection. Despite the high mortality rate in this condition, this patient survived complicating Gram-negative bacteremia, metabolic acidosis, hypokalemia, and insulin resistance. She improved significantly with conservative management, but when antibiotic therapy was stopped clinical evidence of infection recurred each time. The unusually long ileal loop used in this patient to construct a urinary conduit produced recurrent hypokalemic, hyperchloremic acidosis. Ultimately, surgical drainage of the perirenal abscess without nephrectomy produced significant improvement.

Introduction

Emphysematous pyelonephritis and perirenal emphysema are rare conditions in which an infection of renal or perirenal tissue, usually with Gram-negative organisms, is associated with local collections of gas. This constellation is often encountered in patients with lowered resistance and many such patients have been reported who had diabetes mellitus and azotemia. In addition, obstructive uropathy is frequently associated.[1-14] About one-half of such patients die.[13] Treatment has varied from conservative management with antibiotics, to surgical drainage and nephrectomy.

In this communication we report a diabetic woman, with an ileal conduit, who developed perirenal emphysema. This case is of particular interest because the patient survived complicating Gram-negative septicemia, hypokalemia, hyperchloremic acidosis and insulin resistance.

From the Department of Medicine, University of Toronto and the Division of Nephrology, Toronto General Hospital, Toronto, Ontario, Canada.

Case Report

A 36-year-old, French-Canadian woman was admitted in November 1969 for treatment of uremia, severe metabolic acidosis and pyelonephritis with gas collection around the left kidney.

In 1968, she underwent hysterectomy for adenocarcinoma of the endometrium, followed by a course of radiotherapy. In November, a vesico-vaginal fistula developed and attempted surgical closure was unsuccessful. Therefore, an ileal conduit was constructed in January of 1969. A particularly long segment of distal ileum was needed for this purpose because the patient was markedly obese. After discharge, she returned to work for the next eight months. In October she began to have polydipsia, nausea, vomiting, diarrhea and left-sided flank pain. Two days later, after the onset of chills and fever, she was admitted to another hospital where radiographs showed gas surrounding the left kidney and urine culture grew Aerobacter. She was started on chloramphenicol, 2.0 g per day. She continued to deteriorate, became progressively more obtunded with a profound metabolic acidosis with an arterial blood pH of 7.11. Her total carbon dioxide was 9 mEq/1 and serum potassium was 2.1 mEq/1.

She was then transferred to the Toronto General Hospital, and on admission she was profoundly ill, confused and had Kussmaul's respiration. The blood pressure was 170/108 mm. Hg., heart rate 92/min and regular, temperature was 37°C, and respiratory rate was 34/min. There was an ileostomy bag in situ over the abdomen and the left flank was tender. The results of laboratory investigations are shown in Table 1. Her Hgb was 12.6 g, white blood count 27,300/c.mm and the platelets appeared normal. She had no ketones in the urine on admission. Abdominal x-ray films demonstrated a sickle-shaped collection of gas around the left kidney (Fig. 1A and B).

Despite vigorous intravenous fluid replacement, her blood pressure fell and her jugular veins did not fill in recumbency. Her temperature rose to 39°C later in the day. Initial treatment of her acidosis, profound potassium deficiency and diabetes is outlined in Table 1.

Initially her antibiotic treatment consisted of 30 million units of penicillin intravenously in each 24-hour period and

Table 1

Laboratory Data and Treatment

	On Admission	*Day 1*	*Day 2*	*Day 3*	*Day 4*
Arterial blood pH	7.1	7.11	7.35	7.43	7.47
Serum sodium (mEq/)	126	130			
Serum potassium (mEq/1)	2.6	2.5			
Serum chloride (mEq/1)	96	108			
Total carbon dioxide (mEq/1)	5	7	11	18	19
Blood sugar (mg/100 ml)	396	297	348	378	783
B.U.N. (mg/100 ml)	102	99	88	60	42
Creatinine (mg/100 ml)		3.5	2.7	2.3	
		Treatment			
Sodium bicarbonate (mEq I.V.)		396	308	132	
Potassium chloride (mEq I.V. or p.o.)		480 (I.V.)	240 (I.V.)	180 (I.V.)	50 (p.o)
Insulin (units/day)		220	140	150	220

Fig. 1A. Flat film of the abdomen demonstrating sickle shaped gas collection around left kidney.

Fig. 1B. Tomogram of left kidney with perirenal emphysema.

100 mg of gentamicin per 24 hours. Her temperature declined to 38°C and after the sixth hospital day she was switched to 200 mgs of colistin per 24 hours because of the fear of gentamicin toxicity. A blood culture taken on admission grew Klebsiella. After three weeks in hospital, all antibiotics were discontinued and nitrofurantoin was instituted as long-term therapy. Her left-sided flank pin gradually decreased but gas collection persisted in the left perirenal area. Further studies, including aortography, and selective renal arteriography failed to reveal any evidence of renal parenchymal loss or intrarenal abscess formation. About December 9, the patient again complained about headaches and left-sided flank pain and her temperature rose to 39.5°C, while on nitrofurantoin. Urine cultures had yielded growth of Pseudomonas aeruginosa and she was given carbenicillin in a dose of 16 g per day intravenously. Eight days later after her general condition had improved, the left perirenal area was drained through a flank incision. At operation, gas escaped under pressure and the wound discharged a moderate amount of foul-smelling pus which grew E. coli, streptococcus, Pseudomonas aeruginosa and Enterobacter. The wound was kept open with a drain. The E. coli proved to be sensitive to tetracycline which was given for 2 weeks. After three days without antibiotics, her temperature rose again and wound cultures yielded Pseudomonas aeruginosa. Chloramphenicol 1 g per day was given for approximately 10 days. Fever did not recur when the therapy was stopped. She was discharged on long-term nitrofurantoin therapy. During her hospitalization, her hyperchloremic acidosis recurred, and she was maintained on alkalinizing potassium salts.

Discussion

Perirenal collection of gas and emphysematous pyelonephritis are rare and appear to be most common among diabetic patients and individuals with urinary tract obstruction, and lowered host resistance.[13] These patients usually present with fever, vomiting and flank pain. There may be gross pyuria, and bacteriological investigation of the urinary tract infection most commonly yield E. coli, Proteus and Aerobacter aerogenes.[13] On radiologic examination, variable degrees of perirenal gas collection are found and occasionally the renal parenchyma contains gas-filled cystic spaces.[12,13] Tomograms best reveal the extent of the gas collections. Renal arteriograms may be helpful in delineating any unperfused renal segments. Papillary and medullary necrosis frequently accompany the widespread tissue destruction.[8,9,13]

The pathogenesis of perirenal gas collections is not known. We assume that it originates from an area of pyelonephritis or through hematogenous spread. Lowered host resistance undoubtedly plays a significant role and the high numbers of individuals with diabetes and renal failure among patients with emphysematous pyelonephritis constitute indirect evidence for this. Diabetics show decreased chemotaxis and decreased migration of leukocytes,[15] and the higher glucose levels in diabetic patients may provide increased amounts of substrate for bacteria able to ferment sugar and to produce carbon dioxide.[1,8]

Schainuck, et al reviewed the clinical features and operative or autopsy findings of emphysematous pyelonephritis based on 22 cases collected from the literature and one of their own.[13] The overall mortality was 50%. This

can be attributed to inadequate concentrations of antibiotics in the necrotic, gas-filled perirenal space and complicating obstructive uropathy, renal failure and shock. More than one-half the patients had diabetes, and 20% had obstructive uropathy. The authors recommend nephrectomy to decrease the high mortality of patients effected with this condition.

The patient, reported here, appeared too ill on admission to permit any surgical procedure. With aggressive medical management of her various problems, she recovered but developed clinical evidence of uncontrolled infection each time antibiotic treatment was discontinued. After surgical drainage, she had a decisive improvement and nephrectomy was not necessary. On the basis of this experience, we suggest that a conservative approach is justified when gas collection and necrosis are confined to the perirenal area and if renal arteriograms show uniform complete perfusion. Surgical drainage of the perirenal pus collection can then be done as an elective procedure and the kidney may be salvaged.

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REFERENCES

1. Senator, H.: Über Pneumaturie im allgemeinen und bei Diabetes mellitus insbesondere. Internat. Beitr. z. Wissensch. Med. (Festschr. R. Virchow) 3: 317-332, 1891.
2. Goldstein, A. E. and Abeshouse, B. S.: Gas bacillus infection in urology. J. Urol. 31: 547-586, 1939.
3. Alexander, J. D.: Pneumopyonephrosis in diabetes mellitus. J. Urol. 45: 570-578, 1941.
4. Gillies, C. L. and Flocks, R.: Spontaneous renal and perirenal emphysema. Amer. J. Roentg. 46: 173-174, 1941.
5. Winsted, G. A.: Intrarenal gas. J. Urol. 68: 423-429, 1952.
6. Braman, R. and Cross, R. R. Jr.: Perinephric abscess producing a pneumonephrogram. J. Urol. 75: 194-197, 1956.
7. Blum, E.: Les infections urinaires gazeuzes chez les diabétiques. Press Méd. 69: 2238-2284, 1961.
8. Schultz, E. H. Jr. and Klorfein, E. H.: Emphysematous Pyelonephritis. J. Urol. 87: 762-766, 1962.
9. Harrow, B. R. and Sloane, J. A.: Ureteritis emphysematosa, spontaneous ureteral pneumogram, renal and perirenal emphysema. J. Urol. 89: 43-48, 1963.
10. Stokes, J. B. Jr.: Emphysematous Pyelonephritis. J. Urol. 96: 6-11, 1966.
11. Klein, D. E., Mahoney, S. A., Youngen, R. and Schneider, D. H.: Renal emphysema. J. Urol. 95: 625-629, 1966.
12. Yu, S. F.: Spontaneous renal and perirenal emphysema. Brit. J. Radiol. 39: 466-467, 1966.
13. Schainuck, L. I., Fouty, R. and Cutler, R. E.: Emphysematous pyelonephritis. Amer. J. Med. 44: 134-139, 1968.
14. Banks, D. E., Persky, L. and Mahony, S. A.: Renal emphysema. J. Urol. 102: 390-392, 1969.
15. Mowat, A. F. and Baum, J.: Chemotaxis of polymorphonuclear leucocytes from patients with diabetes mellitus. New Eng. J. Med. 284: 621-627, 1971.

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The Need for Certificate of Need Authority

Towards Building a Model Health System for Maine

JOHN W. NORTON*

INTRODUCTION

If a moratorium on health facilities construction were called today, the effect would be a slower rise in health care costs with no adverse effects on mortality/morbidity statistics. Many areas of Maine have too many hospital beds with resulting low occupancies and high overhead expenses. The costs to construct and equip a hospital bed ranges from forty thousand (40,000) to sixty thousand (60,000) dollars and it costs nearly two-thirds as much to maintain an empty hospital bed as a full one.** Rising prices of health care can be related to capital expansion programs going unchecked in many communities, therefore many (twenty) state legislatures have granted authority to stop unnecessary construction. These regulations are called Certificate of Need Authority.

Certificates of Need are awarded to institutions delivering a range of medical, social and educational services to a defined population where there is demonstrable need for these services. In past years, the medical system has reacted to "felt-needs" rather than defined-needs and in too many instances has duplicated existing resources and wasted scarce medical personnel. These facilities have failed to adopt a regional point of view in place of the self-centered "we have to be all things to all people" approach to medical delivery.

Health care will soon be the nation's largest industry, consuming sixty-three (63) billion dollars in 1969 and more than seventy (70) billion in the early 1970's. In the decade of the 1960's, health care costs grew at a rate three times the consumer price index with forty percent (40) of the revenue going to hospitals, twenty percent (20) to physicians and eleven percent (11) for drugs. The single most important contributor to the runaway inflation of the late 60's was the passage of Medicare and Medicaid legislation. The response to the invitation to receive health care for the categorical poor and aged has been overwhelming. Hospitals and nursing homes receive fifty (50), and seventy (70) percent of their income respectively from governmental sources. The relentless increases in Title 18 and 19 appropriations year by year raises the question that if quality health care is the right of every citizen then health care facilities may have to be public institutions.

State legislators by and large are looking to Washington for direction rather than retaining the health care initiatives for themselves. It seems that many health care institutions are waiting for directives from Washington rather than working with the state legislature to develop solutions to Maine's health problems.

The entire area of health care legislation is fraught with rippling effects that can reach tidal wave proportions unless the legislation is carefully researched and specifically written. Health planning professionals when suggesting a change in the system, have to look closely at how their plans might impact:

1) Costs
2) Accessability – Acceptability
3) Comprehensiveness; and
4) Accountability

Any attempts to deal with one of these variables separately frequently has disastrous effects on the system as a whole. Attempts to hold down costs must at once insure that the entire service population can gain access to the service, insure that the range of needed services remains sufficient and that the quality is high with adequate fiscal accountability.

The first responsibility of Comprehensive Health Planning (CHP) is to define the system and attempt to measure how well it is working against some standards. But our job goes beyond defining the areas of needs, uncovering the gaps in the service, pointing out duplication or facilities or the mal-distribution or personnel; CHP must search for ways to implement solutions to these problems. The methods of defining problems and recommending solutions included evaluating existing programs where appropriate, proposing alternatives, identifying needs and ranking them on priority scales, proposing new services and finally activating interested parties to work in these areas.

It should be noted that planning stops short of actually delivering services. This is a very important aspect of the partnership-for-health legislation creating Comprehensive Health Planning. There is a body of health planning professionals that equate planning the health

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**Walter J. McNerney, President of Blue Cross Association, speech 1971.

The Community Hospital

Its Possible Role in Medical Education

BRENDAN CLIFFORD* and JOSEPH JORIZZO**

Pre-medical students tend to enter their respective medical schools rather sure of themselves and of their abilities. For them, the four years of college serve not only as a training ground for the novice physician but also as a hot-house of academic competition. In a very Darwinian sense, the student feels that the fittest have survived and that anyone who pulled all those organic chemistry A's back in undergraduate days couldn't possibly have any difficulty palpating a spleen. He is intellectually and theoretically oriented, expecting medicine to consist of the simple memorization and regurgitation of facts.

Because of this attitude, the first year of medical school provides a rather rude awakening for this well oiled and much vaunted intellect. His bubble is burst. Millions of facts, hard cord pieces of pure data are thrown at him just as they were at college, but now there is a critical difference, it's no longer feasible to just study for tests and then erase the old memory banks. Everything is important; everything must be retained (a Herculean task which is of course impossible). The problem is further complicated by an obscure jargon and the language of medicine is made even more perplexing because there is very little solid material to hang it on. That is, clinical correlations are necessarily scanty, for diseases as they exist in the pathology textboook – pages 811 to page 813. Real people don't have them.

When discussing biochemical pathways, the most obscure clinical entity must necessarily be touched upon creating often a false statistical image in the mind of the student. Idiopathic autoimmune diseases are a dime a dozen while a sore throat becomes an obscure, exotic entity never encountered in any preclinical discussion.

The freshman year closes then on a perplexing note; the student's opinion of himself is shattered; he's simply not as smart as he thought he was; his concept of the pathological universe is necessarily, albeit unfortunately, distorted as is his picture of the patient – that nameless and faceless creature occasionally encountered at a clinical pathological correlation.

All who have studied medicine can attest to the psychological vacuum in which the preclinical medical student dwells. We feel, however, that we have been fortunate enough to find a possible solution to this problem. Interestingly enough the crux of that solution rests in the hands of that infamous whipping boy of all CPC's, the L.M.D. – that same physician who still manages to filter out and successfully treat 80 percent of all clinical problems.

Under the SAMA program, and financed by the RMP program of Maine, we were both able to spend approximately two months with a rural G.P., Dr. Charles H. Lightbody of Guilford, Maine. In being glued to his heels, we learned something about that physician to whom the city based academician renders lip service but really doesn't understand, the general or family practitioner. Our mentor has impressed us as approaching the old-fashioned ideal of the selfless, tireless, well-rounded physician. He and his five colleagues, all solo G.P.'s utilize the Mayo Memorial Hospital in Dover-Foxcroft, a thirty-five bed hospital providing facilities for an area encompassing some 15,000 people. Collectively they enabled us to sample community medicine at the grass roots and emphasized for us the human side of practice; the art of medicine, which struggling students often times find hard to learn and so easy to forget.

Our preceptor was not what we expected; he was really not a generalist but rather a semi-specialist in five separate fields; internal medicine, surgery, obstetrics, pediatrics and psychology. In following such a varied practice and pursuing a carefully planned rotation with other physicians, hospitals, and health organizations, we were able to get a taste of the variation and diversity of rural medicine at it exists in that area of the State.

Dr. Lightbody's personality as well as his practice had a special, even perhaps crucial impact on us at this point in our medical education. Pre-clinical students need a mentor to identify with, a physician who can make concrete the art of medicine and yet make it a viable thing for the novice. The conclusion of the freshman year of medical school is a particularly impressionable period; one might even say a critical period. The initial disillusionment and self depreciation of this first year can be overcome in the picture of a man who has a working knowledge of medicine.

No – every page doesn't have to be memorized after all; as you use it, it comes back. No – lupus and gargoylism aren't so prevalent after all, but no two inflamed throats look alike. No – the doctor isn't just another technician. He is a respected and integral part of the community.

The community – this brings us to one unique aspect of medicine which Maine has to offer. Driving through the Maine countryside, one might get the impression that trees are its most important resource. Nothing could be further from the truth. Although in relatively short

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supply, people are Maine's greatest asset. We have both found them incredibly open and alive, in love with life. But on a more pragmatic level, rural Maine's distinctive type of community life is a gold mine for the medical student. A young doctor in an urban medical school is trained to interview a patient he never met before, take a history he never can confirm or corroborate and at the same time treat this patient with the near certain knowledge that he may never see the fruits of his efforts; that patient might never show up again.

In rural Maine, all these problems are non-existent for everyone knows everyone else. Social histories aren't essential. Family backgrounds and working conditions do not have to be explained; everything is known. A student trained in the fine art of "history taking" and "personality guessing" finds his perfect testing grounds here and it's amazing how often these same educated guesses find themselves wanting when exposed to the cold hard light of the family physician and his firsthand experience with a particular patient. Thus, even if the student merely concentrated on interviewing technique, he would find his time in Maine well spent.

This close knit type of social structure helps buoy the spirits of the pre-clinical student in another way, the recovered patient just doesn't disappear. He stays in sight and usually successfully re-enters the community.

There is another thing that Maine didn't let us forget, as the license plate indicates, there is another side to life. It is truly vacationland. This summer our many hosts have taught us how to relax proving that there is a time to work and a time to play and the latter takes skill and practice also.

One important spin-off of our type of experience could well provide at least a partial answer to postgraduate medical education for practicing physicians. Formal courses, TV and radio programs and other typical efforts to provide such education have been notoriously unsuccessful since they have difficulty in attracting and keeping the doctors attention. It is our feeling, along with that of many practicing physicians that medical education in the community hospitals in a format somewhat similar to what we have experienced would be a great boon to the community and its doctors as well as being beneficial to the student. For it is a truism, one must teach in order to learn. Perhaps the greatest service that students can render is the impetus they provide for their mentors. Most lay people in a community are not aware of the incredible investment of time and energy required of physicians in order to keep abreast of the rapid changes in the field of medicine. Most physicians agree that the single, most powerful tool for this continuing postgraduate medical education is teaching. The physician-teacher gains back directly and indirectly almost as much or more benefit than does his student. The student by definition is a questioner. The physician-teacher upon being questioned is forced into intellectual stimulation by mentally reviewing concepts and arguments that may be on the verge of being forgotten. Frequently one may think that he has complete grasp of a concept but only when asked to explain what he knows about a subject does he realize that there may be several weak links in his arguments.

Thus, by teaching, one has a chance to gather his thoughts on a topic and to re-evaluate his feelings on a given subject. One may also be forced to admit to himself that his mastery of a given area is not complete or nearly as extensive as he himself thought. It is important that the student ask basic questions. These are the questions that colleagues do not ask one another. Frequently these elementary basic facts, the foundation of ones medical knowledge are easily forgotten. Sometimes a question sets mental gears in motion and this in itself is enough to recall the facts that bring ones knowledge of a subject back to par. There are other times when this is not the case. During these times, the students' questions have a second benefit for the physicians' continuing education. A physician is forced, out of a commitment to his student, his ego, and his own education, to consult the medical literature. He may even be motivated to broaden his reading of the literature in order to be prepared for future questions. The physician-teacher may also expand his reading so as to be able to suggest appropriate articles for his students to read. Naturally, pre-clinical students from the first two years of medical school do not have much knowledge to contribute directly to the physician, but perhaps an occasional tidbit of applied basic science could be offered and might assist a physician in treating a patient. Sometimes the mere questioning of a procedure by a young student may influence a physician's decision on the treatment of a patient.

Closely related to the issue of continuing education is another benefit attributable to students. When a young student is pursuing a busy community physician, the physician is usually intent on imparting to the student some knowledge of good medical technique. Historically, in the Hippocratic tradition, the physician is obligated to teach aspiring young physicians. This duty is fulfilled whenever the physician-teacher makes a sincere effort to show the student good, thorough technique. This benefits both the physician and the patient for it is frequently very helpful to re-examine some of the shortcuts that the physician may have been accumulating over the years. In short, it may be that the young pre-clinical medical students can benefit local physicians and indirectly benefit the entire medical community in several areas of postgraduate physician education; the pre-clinical student serves as a strong goad, providing a continuous force, fostering physician education, re-education, re-examination, and refreshment.

Another hopeful byproduct of the SAMA-MECO program might be the attraction of new physicians into a given community. The principal way to bring new people into an area is to expose these same people to the area. Earlier we discussed the eagerness exhibited by the pre-clinical student to correlate his newly learned concepts with real people. Maine is especially beautiful in sum-

mer. Most pre-clinical students still have the summer vacation between their first and second year. What a perfect opportunity to mate the interest of the student and the community. Here the State has a chance to offer invaluable clinical and sociological community medicine experiences to medical students at a time when students most want and need it. The young student may well be bitten by the bug of family practice in Maine and return to the State.

It is our impression, after discussing this matter with a number of medical students working in the State this summer, that their experiences were overwhelmingly positive. Even if these students do not eventually return to practice in Maine, the State will have at least gained goodwill ambassadors, for the grateful students will attempt to find ways to express their thanks for being given a beautiful experience at the perfect stage of their training.

The profession in Maine is well aware of the desperate need for more family physicians. Upon meeting most generalists in Maine one is struck by a fascinating duality. While they radiate exuberance, energy and dedication, if one looks a bit more deeply, he can see the beginnings of a fatigue that is the result of years of tremendous overwork. Waiting rooms are overflowing and the number of hospital outpatients grows daily.

It would seem time for communities in this State and elsewhere to become more aggressive in their efforts to deal with this physician shortage. Waiting until the problem becomes completely overwhelming is not valid. Programs such as the Regional Medical Programs – SAMA-MECO, which brings pre-clinical students into the State during the summer are steps in the right direction. Hopefully with the expansion of facilities through the regional hospital concept, rotating medical training programs will be developed at the externship, internship and residency levels. If these programs are oriented towards general family practice, an area where Maine has a great deal to offer, much may be gained in the effort to provide the State with needed physicians.

There is a third way students can benefit the community. The young student can sometimes provide objective, fresh insight into some community problems. The pre-clinical student can and should become involved in the social life of the community. After all, medicine is people. To be able to understand their health problems, one should be aware of the environment in which the people live. The term environment connotes physical, social and psychological surroundings. Awareness of the community in all its different facets is a necessity. The pre-clinical student who is learning at a community hospital should be exposed to the community as well as the hospital. The young student will almost certainly become involved in many community issues, especially those concerned with health. At service club meetings, hospital staff meetings, community planning meetings and other local activities, the student can frequently offer an objective and different view point. Since the student is by definition a learner and since he is only a temporary resident, he may be able to offer criticisms that others might be loath to offer. He is in an ideal position to contribute his ideas to such a gathering. In the light of the advantages to both student and community, it is hoped that the role of the community hospital in medical education will be an expanding one.

In summary, we would like to stress that although our summer experience with the doctors and patients of Maine would be beneficial to any student in medicine, it is our opinion that it would be particularly valuable for the impressionable pre-clinical students, those who are equally fascinated by the small things seen in practice as by the grandiose and dramatic, those who are as interested in seeing a T&A performed as they are in watching neurosurgery. It is at this level in their career that future doctors are most impressionable, when they can most readily identify with a mentor (perhaps even idealize him). It is in this pinpoint of time at which they can catch that contagious love of life that the art of medicine has to offer.

SUMMARY OF REPORT

by James H. P. Garnett, M.D. and Joseph L. Hasenfus

The Student American Medical Association Project for Medical Education and Community Orientation (SAMA-MECO), a nationwide program, had its introduction into Maine in the summer months of 1972; providing an educational program for ten pre-sophomore medical students. Maine's Regional Medical Program provided the major portion of financial support required, plus the necessary staff activity for the overall coordination of the project. Advisory and cooperating organizations and individuals were: Maine Medical Association, Maine Osteopathic Association, Maine Hospital Association, Maine Academy of Family Physicians, the respective hospitals involved, and the local physicians directing the programs of the individual medical students.

As expressed in narrative reports and in discussions, the consensus of the students involved was outstandingly affirmative regarding the various values of the summer experience for them. Five of the seven physicians directing the activities of the ten students have responded to a post-program questionnaire; all rating the project highly and indicating interest in participating again.

In addition, an eleventh pre-sophomore medical student was involved similarly in this summer program in Maine but not enrolled in the SAMA-MECO Project because of the specialized nature of his assignment with the Maternal and Child Health Program at Ellsworth. Interestingly, his narrative report was quite like those of the others and indicated strengthened interest in primary medical care.

A Medical School For Maine: The Commitment of the Maine Medical Association

Richard T. Chamberlin, M.D.*

Introduction

The concept of developing a medical school in Maine is not a new one. Previous efforts were successful from 1820 to 1921 at Bowdoin College, and from 1828 to 1833 at Colby College. There was an unsuccessful attempt in 1881 at the Eclectic Medical College of Maine in Lewiston. History also records that in 1951 the Maine State Legislature authorized the University of Maine to operate a college of medicine, and in 1960, the Council of the Maine Medical Association voted to support a two-year medical school program in Maine. Although this effort failed because of lack of funds to support a needed feasibility study, such a study was finally completed in 1966 by John Truslow, M.D., with the conclusion that a medical school undertaking was not feasible.[1] Medical Care Development, Inc., Maine's Regional Medical Program, and its staff has made a thorough study of the successes and failures of the past, and has placed them in proper context with the present day needs for medical manpower. The results of this study and its recommendations were published in December of 1971 in a report entitled, "A Rationale and Program For A College of Physicians In the State of Maine." Some of the basic recommendations of the report have been previously published in this journal.[2] Out of this study grew a new concept for a medical school – a concept so innovative that it is important to rethink previous conclusions about establishing a medical training program in Maine. It is important to stress that previous feasibility studies are not necessarily applicable to this present concept.

Although the Maine Medical Association had been involved in some of the early planning for the new medical school, and had assisted in the formation of a group of physicians to act in an advisory capacity to the Chancellor of the University of Maine, there were many physicians who were either unaware of the details of the proposed new school, or if aware were opposed to the concept. The Maine Medical Association had never officially voted its full support to the most recent project, although it had accepted progress reports from Maine's Regional Medical Program at each of the annual meetings of 1970 and 1971.

Discussions at the Executive Committee level in January 1972 concluded that in spite of the previous information and reports, the physicians on the committee did not fully understand the development well enough to make an informed recommendation. Although there were many questions raised, the two general areas of the innovative program recommended by Maine's Regional Medical Program which caused the most concern were those involving the use of the problem-oriented curriculum in medical school teaching and the use of the community hospital as opposed to the university hospital as a locus for the clinical training of the medical students. Both of these areas overlap and involve other controversial areas such as changes in admission requirements, student directed learning, the use of automated audio-visual techniques in the learning process, and the place of basic sciences in the curriculum. The Executive Committee of the Maine Medical Association decided to study the problem further.

Method

In the effort to gain better understanding of these new concepts, the Executive Committee of the Maine Medical Association appointed a group of physicians to visit schools that were now using some of the techniques described in the Medical Care Development Report. It was also suggested that each physician visit his own medical school if he had not done so recently, to learn what changes might be occurring in the older, well-established schools everywhere, changes which may have escaped the recognition and knowledge of the busy, practicing graduate of such schools.

The study group selected included three men in family practice, two in internal medicine, and one surgeon and was fairly well representative of the State geographically. In addition, the group consisted both of those who were already favorably inclined to the medical school development, as well as those who were quite opposed to the plan as being feasible.

The visit included Michigan State University of Human Medicine, which among other things is using community hospitals entirely as the site for clinical training of their medical students, and the Medical College of Georgia, which has an innovative program on an experimental basis using an entirely new curriculum approach which has been called the problem-oriented curriculum. Other schools visited by individuals of the study group included Tufts University School of Medicine, St. Louis University, the University of Virginia and Cornell University. Upon return, each member of the group was asked to submit his own ideas and impressions in writing. From these written reports, a formal report was submitted to the Interim Meeting of the House of Delegates of the Maine Medical Association and parts of that report are included in the next section of this article.

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*Thayer Hospital, Waterville, Maine 04901.

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Descriptions

Michigan State University College of Human Medicine – Michigan State University College of Human Medicine began as a two-year medical school in 1966 and is currently expanding to a four-year program graduating its first class in the spring of 1972. There is no university hospital at the Michigan State University although one is planned in the future. The visit to Michigan State University included a series of discussions with Ronald Richards, M.D. of the Office of Medical Education; Donald Weston, M.D., Associate Dean; Andrew Hunt, M.D., Dean; Dr. James Thomas who is Professor of Anatomy and who currently was also a fourth-year student at the medical school; and Thomas Johnson, M.D. of Grand Rapids, Michigan who is a physician who is currently a director of a community hospital program in the Michigan State University organization. Finally, discussions were held with two fourth-year medical students who were about to graduate from the program in the first class.

Dean Hunt summarized the basic approach used at Michigan State University. He stated that they were trying to answer the question, "What does a medical student need to know to make an appropriate and correct medical decision and then where can these things be learned best?" Dr. Richards outlined the program as follows. It should be noted that it is difficult in these days of curriculum reform to identify the traditional semesters or years since the time slots are divided in a slightly different and more flexible way than in traditional medical schools.

(1) Entry Profile. This consists of a brief period of time at the outset of the medical training and consists of a series of tests and evaluations of the students themselves in an effort that the students may understand their own capacities as well as that the college may learn more about the students in ways other than purely academic. Certain psychological tests and types of aptitude evaluations are included.

(2) Phase I. This consists of an introduction to medicine and medical care. This is done in small groups in the community and includes lectures, presentations of what are termed focal problems. These are picked both for their medical content as well as for their implications for medical problem solving. There is also an introduction to the doctor-patient relationship using simulation techniques.

(3) Phase II. This consists of a block of time during which basic sciences are taught. The focal problems are presented in more detail and students participate in a continuity of care clinic. During this time, tests are administered to the students giving only a pass/no pass type of result. At the end of Phase II, which is approximately at the end of the second year, the student takes a primary clerkship of ten weeks in a community hospital. In these clerkships, a problem-oriented medical record approach is used and team teaching is done.

(4) Phase III. This consists of a specialty clerkship period. It is approximately two years in length. The student is encouraged to define for himself what he wants to accomplish by the end of his program and to design for himself which clerkships will best accomplish this goal. There are, however, a certain number of mandatory clerkships.

The hospitals used in the Michigan State University Program are located in hospitals who volunteer to be in the program. The hospitals range in size from one hundred and thirty-six beds to six hundred and fifty-seven beds with an average size of three hundred beds. Some have a full-time director of medical education but none have full-time chiefs of services. In most communities, the program is financed by funds channeled through an educational foundation.

The two students who were interviewed during the course of the visit were very enthusiastic about the program that they were just completing and both were going on into a family practice residency sponsored by one of the hospitals in East Lansing, Michigan. Perhaps the best look at the program, however, came from Dr. Johnson who is the Director of one of the community hospital programs. He honestly detailed some of the many problems encountered on a day to day basis, such as difficulty in getting commitments from the staff to teach and difficulty with interrelationships between the community hospital and the medical school. He emphasized, however, that the program can work very well if one identifies an energetic and interested man at the community hospital level to be the local coordinator. It is emphasized that this person is not necessarily completely involved in teaching but is involved in coordinating the student's program, securing the teachers from the staff, and in general seeing that the students' stay at the community hospital is a dynamic one.

Medical College of Georgia – The Medical College of Georgia has been under pressure recently, as many medical schools have been, to increase the size of their entering class. They had done this on two occasions in recent years and had reached absolute capacity – that is, it was physically impossible for them to add another student or another desk to much of their physical plant. On further insistence of the State Legislature to increase the class still further, and with a commitment from the legislature for further traditional medical school construction, the faculty at the medical college asked and secured permission from the legislature to conduct an educational experiment that would allow an increase of the class an additional ten percent and yet to do this outside the presently existing overcrowded facilities.

Upon securing this permission, the medical school constructed an inexpensive prefabricated building including some large classrooms, some individual teaching carrels, and having strong emphasis on audio-visual materials such as tapes, closed circuit television, video tape, as well as a library and dissecting facilities.

The students for the experimental program were selected by an interesting process. The entire freshman class was notified by mail that an educational experiment

was about to be conducted. Of the large number of volunteers that resulted from this initial announcement, a class of sixteen was to have been selected. The volunteer group was subjected to a process of random number identification and the sixteen were selected on the basis of these random numbers. Thus, there were three groups resulting from the initial freshman class at the Medical College of Georgia. The first group were those who didn't volunteer at all. The second group were those who volunteered but were not selected. The third group were those who volunteered and were selected. The college is interested in studying these three groups as they proceed through their medical training to see if the parameters to evaluate the students in the experimental program can be clarified. In the first experimental program, the sixteen students will be absorbed into the third-year class and graduate as usual with their entering classmates.

The visiting consultant group met with William Chew, M.D., Director of the experimental program, and Robert G. Crounce, M.D., Associate Dean, School of Medicine. A meeting was also conducted with several of the medical students in the experimental program as well as with a number of the other members of the faculty.

The Program – The program itself is constructed around the problem-oriented curriculum approach. Rather than teaching the traditional basic sciences in segments such as anatomy, bio-chemistry, and physiology the student is led to these basic science subjects via the presentation of patient problems. These presentations are typical case presentations and are drawn directly from the records of the university hospital. They are presented in typical medical teaching fashion except for the fact that at the end of the presentation the teacher lists the problems or helps the students develop what problems the patients have presented. One of these problems, for instance, gout would then lead into such basic science teaching as the intermediary metabolism of purines, the pathology of gout, etc. The study group of physicians from Maine sat in on part of a day's lecture that was being conducted in this particular fashion. Later these students would be led to the hospital where they would see patients with gout, have an opportunity to examine uric acid crystals under the microscope, and in general see the relevance of some of the basic science material in a clinical setting right from the start of their training. The teaching of anatomy is being done by the use of plastic models, and audio-visual techniques, although there is a cadaver present and dissection is carried out by a prosector for the students as needed and as frequently requested by the students.

Evaluation – It is obviously too soon for a complete evaluation since the end of the first year has just been completed. Thus far, a type of examination similar to a national board examination has been administered to the entire freshman class and the students of the experimental program have ranked very well in relation to their classmates.

The faculty in the Medical College of Georgia is impressed enough by this approach that in the coming year a second experimental program is being planned. In the new experiment, an entire class will be taken through three years of medical training in the fashion described above with some modification along the way. The new director of this program explained that he has been able to evaluate some of these new methods and has been impressed by their effectiveness. For instance, he personally has taught one hundred and eighty-six students an entire histology course in eight separate laboratories using transmission over color TV only. In an evaluation, these students did ten percent better than students taking the traditional program. The faculty members working in this program in Georgia feel that they will be able to produce a "package teaching program." This package program of medical education will include modular units which would be quite mobile, rely on self-instructional aides, and literally could be taken into any part of the state where they might be necessary not only for medical education on the undergraduate level but also for continuing medical education.

Summary

Upon return from the consulting trip, the members of the Executive Committee who participated individually wrote their impressions and recommendations. From these the following consensus, conclusions, and recommendations were offered to the Executive Committee and to the House of Delegates at the Interim Meeting at Waterville in April of 1972.

Conclusions

(1) The community hospital is a logical place and an effective locus for the clinical teaching of medical students in all levels of their training. The success of such training depends on the identification of a strong local program coordinator and the use of well-motivated practicing physicians in the community as teachers.

(2) The use of a focal problem or problem-oriented curriculum design allows for the efficient and effective teaching of basic sciences in a way which is more relevant to clinical problem solving.

(3) The problem-oriented curriculum design provides the student with a self-determined methodology which may be used in a lifetime of continued learning.

(4) The plan for a medical school without walls is viable and relevant to Maine's needs.

Recommendations

(1) Further efforts to acquaint the people in Maine with the innovative suggestions about a medical school for Maine should take place within the State. Specifically, a symposium should be designed to reach not only physicians but all legislators and educators within the State. The faculty for this symposium could be drawn from any

number of institutions, but particularly the Medical College of Georgia and Michigan State University have offered their assistance.

(2) The University of Maine should hire a Dean of the Medical School as soon as possible and further developmental work and planning be implemented through the University with the assistance of Maine's Regional Medical Program and the Maine Medical Association.

(3) The basic structure of the medical school program should be the problem-oriented approach.

(4) A basic science faculty capable and willing to teach in this kind of curriculum design should be recruited. It is the opinion of the study group that this recruitment might well have to take place outside of the State of Maine rather than relying wholly on the existing basic science faculty in the current University of Maine system as proposed in the Rationale and Program For A College of Physicians In the State of Maine.

(5) The plans for a medical school without walls should be considered so viable and important to the physicians and the people of the State of Maine as to warrant official active support by the House of Delegates of the Maine Medical Association and every physician in the State.

As a result of this report and these recommendations, a resolution was prepared and submitted to the House of Delegates at the Annual Meeting of the Maine Medical Association on June 11, 1972 at Kennebunkport, Maine. The resolution was adopted unanimously by the House of Delegates. The resolution in its entirety is stated as follows:

WHEREAS, Medical Care Development, Inc. with its advisory committee of nineteen physicians has developed and released a report entitled, "A Rationale and Program For A College of Physicians In the State of Maine," which recommends the establishment of an innovative medical school without new walls in the State, and

WHEREAS, the University of Maine has placed a high priority on the study of the report and its recommendations including the obtaining of consultant services from Dr. Oliver Cope, and

WHEREAS, the Executive Committee of the Maine Medical Association has studied some of the aspects of the proposal in depth using the method of a special physician group consultation team which visited and observed similar innovative medical school programs, and

WHEREAS, the report of this consultive group was favorable to the project and recommended that positive action be taken by the Maine Medical Association, now therefore

BE IT RESOLVED, that the House of Delegates of the Maine Medical Association recommend that the University of Maine should continue to develop the concept of a medical school for Maine with all deliberate speed, and further

BE IT RESOLVED, that the Maine Medical Association pledge its active support to the University of Maine in this development.

Discussion

Any approach to the problem of medical education must be made cautiously. Just as the scientific medical literature has exploded in volume in recent years, so too have the writings in the area of medical education at all levels flourished.

This report is not meant to be a thorough review of the literature on the subject. There are, however, some points which bear emphasis in relation to the concept of a medical school in Maine as defined in the Medical Care Development Report.

The goals for the proposed medical school provide a thought-provoking place to start. No thought has been given to the idea of establishing a medical school for the mere prestige of doing so. Rather, one of the primary goals of the proposed school is to establish an educational experience whose emphasis is family practice.[3]

In this regard in many recent discussions about the proposed medical school one frequently hears the question – "But is there a need?" One way to answer this question might be to look at the need for primary care physicians in the State. One study concluded that one hundred and thirty-three primary care physicians are needed for each one hundred thousand population, if these primary care physicians are to provide high quality work as defined by well accepted criteria.[4] Translating this estimate to Maine's nine hundred and ninety-three thousand population would indicate a need for about thirteen hundred and sixteen primary care physicians in the State. Allowing for certain recognized problems in definition, the best current estimate of primary care physicians in this State currently is seven hundred to seven hundred and fifty. Thus, we have an estimated deficit in primary care physicians. How is this deficit to be met? Can this new program meet it?

To answer these questions, one must obviously study the current potential supply of new physicians. In 1971, there were ninety-four students from Maine in thirty-two medical schools in the United States and Canada. Even if all of these were to elect family practice and return to Maine, which is highly unlikely, they would add only an average of about twenty-four new primary care practitioners per year. Even allowing for adjustments made necessary by the natural attrition rate of physicians, and for the fact that students currently in medical schools from other states may well settle in Maine, it is difficult to imagine existing programs, even if expanded, meeting the needs as expressed by these data.

The report of Maine's Regional Medical Program also explores in depth all of the alternatives available to increase the supply of new physicians. These include cooperative arrangements with existing medical schools, physician expander (physician's assistant) programs, internship and residency programs, preceptor and clerkship programs, enticement methods, and a two-year basic sci-

ence program.[5] Any or all of these programs obviously have their merits but none singly or totally are felt to be sufficient to meet Maine's needs as defined by the broad goal as stated – to – "improve the health care of the people of Maine through expanding the capacity of the provider community for both quality and distribution of services."[6]

One logical next question is, "Will the proposed school, if successful in starting, answer the needs as expressed by various studies?" Obviously no one can know this answer for certain. Those members of the study group who were the most doubtful before the study were convinced that the elements of the proposed program could be successful in the State of Maine. The feelings of the entire group were perhaps well summed up in a letter from Robert G. Crounce, M.D., Associate Dean, School of Medicine, Medical College of Georgia, which was received after the group returned home. He said in part, "Somewhere in this country, some year soon, will be established a true 'school for health careers.' This school will include educational opportunity for a variety of health careers at various levels of performance, criteria of intellect and of competence. Some of the graduates will be practicing physicians, some physicians' associates, some pediatric nurse practitioners, some dental hygienists, some laboratory technicians, some health career officers. The pathways through this school will be multiple, will offer alternatives both in pathways and in time, and will allow crossover between ladders or ascension of ladders dependent upon successful performance in terms of attainment of prespecified behavioral objectives. This school will be exciting, difficult to manage, eminently fundable at the federal level, and will generate hostility and opposition. Its reward, if successful, should be more than sufficient to balance the difficulties. Since this school will begin somewhere, some year soon, why not in Maine?"[7]

Although not all of the ideas expressed in this paragraph are included in the Medical Care Development Report, the report is just as innovative. Those who have been in opposition to the medical school development have seemed to be so as if the goals of the medical school were those of a traditional school. It is suggested that all should obtain and read carefully the entire Medical Care Development Report to learn that what is being proposed is an entirely new concept of medical education and one which has been heartily endorsed by the entire study group appointed by the Maine Medical Association as well as by the House of Delegates of the Maine Medical Association.

REFERENCES

1. Medical Care Development, Inc., 295 Water Street, Augusta, Maine, 04330. "A Rationale And Program For A College Of Physicians In The State Of Maine," Exhibit 4, pp 42-46, December 1971.
2. Blewett, E. Y., EdD., and Chatterjee, Manu, M.D., "Health Science Education: Medical School Component." Journal Maine Medical Association, 62: 71-72, 77, 1971.
3. Medical Care Development, Inc., Ibid, pp 2, 3.
4. Schonfeld, H. K., Dr. PH: Heston, J. F., MPH, and Falk Isidores, PhD., "Numbers Of Physicians Required For Primary Medical Care." New England Journal of Medicine, 286: 571-576, 1972.
5. Medical Care Development, Inc., Ibid, pp 16-20.
6. Medical Care Development, Inc., Ibid, p 2.
7. Personal Communication.

THE NEED FOR CERTIFICATE OF NEED AUTHORITY — *Continued from Page* 8

system with controlling the health system. They want regulations that will give teeth to the organization and clout for its recommendations. It is their contention that Certificate of Need Authority legitimately belongs with CHP. Their argument is that if CHP is in the business of defining needs, then it should additionally have the authority to implement plans.

In Maine we consider the health care system as a giant industry that can benefit from CHP direction: direction that management can use in making rational decisions. CHP defines needs, outlines programs, suggests management goals and objectives and gives indications of overall direction and progress of the industry. Planning is an essential component of management, it is not a control function, but is related to it. CHP was founded on the principle that cooperation between the providers of medical services and the consumers of medical services could be effected on a voluntary basis.

Locating Certificate of Need Authority within Comprehensive Health Planning in Maine would amount to an admittal that this cooperative effort has failed. We see no indications of this in Maine. CHP in Maine is the force for planning the system, not running it.

Once again, we view Certificate of Need Authority as an important mechanism for deterring unnecessary health-facilities expansion. But this power must be vested in the proper hands with careful consideration of its desired effects. Recent hospital licensure regulations from the Health & Welfare Commissioner's office contain some of the elements for Certificate of Need Authority. Although these regulations have not been tested in court, the necessary legislative authority for Certificate of Need could easily be provided to this body by the enactment of enabling legislation.

Under this system, a health facility would request permission to expand a service or construct a facility. Comprehensive Health Planning would be free to represent planning interests to this authority from the vantage point of how this proposal would affect the region or the entire State. Hopefully the institution's proposal would have previously been coordinated with area-wide health planning and differences of opinion, if existing, could be reviewed by the Certificate of Need Authority. CHP views Certificate of Need as the first step towards franchising services, *i.e.,* a public utilities approach to health service delivery. In this system CHP can best serve the interests of all concerned by remaining objective, uncompromised, and capable of planning a system where "the whole is greater than the sum of its parts," a cliché that is certainly not true today.

REFERENCES

Medical Economics, Nov. 8, 1971, pp. 126.
See for example Hospitals, Dec. 16, 1971, Vol. 45, pp. 54-58.
See for example: a) U.S. Dept. of Health, Education & Welfare, Social Security Administration, Social Security Bulletin, Sept. 1969; b) Our Ailing Medical System, Harper & Row Publishers, New York, 1970; and c) Ibid.
Public Law 89-749, Dept. of Health, Education & Welfare, 1966.
Hospitals, Dec. 16, 1971, Vol. 45, pp 51-53.

Rehabilitation Services to Maine's Handicapped Citizens

STANLEY L. BROWN*

In December 1969, the 104th Maine Legislature passed a new Public Rehabilitation Act that reorganized and integrated public rehabilitation services to the State's physically, mentally, emotionally, and socially handicapped citizens.

It did this by establishing a new and distinct unit of public rehabilitation services in the Department of Health and Welfare. This unit was designated by the Commissioner as the Bureau of Rehabilitation, and into it were integrated the Division of Vocational Rehabilitation – formerly in the Department of Education; its adjunct, the Disability Determination Division, which provides a contractual service for the State under the Social Security Administration and operates entirely with Federal funds; the Division of Alcoholism Services, and the Division of Eye Care and Special Services, including its Education, Training and Rehabilitation (ETR) component.

Vocational rehabilitation – the process of helping disabled men and women overcome their handicaps and reach their highest potential of useful, productive living – is either explicit or implied in several of these other Divisions as well as the one that bears this name. It is the objective, for example, of the Eye Care Division's rehabilitation program for working age persons who are vocationally handicapped by blindness or a lesser degree of visual impairment.

Similarly, ETR services are geared to men and women who are socially and economically handicapped and disadvantaged; and Alcoholism Services are addressed to the vocational as well as treatment needs of the clients they serve.

The Division of Vocational Rehabilitation is concerned primarily with helping handicapped persons prepare for and enter into suitable employment. Maine's program of vocational rehabilitation more than 50 years ago, in response to Federal legislation of 1920 – the Smith-Fess Act – authorized funds for joint State-Federal programs of civilian vocational rehabilitation.

Since then, the national Vocational Rehabilitation Act has been amended and revised several times. Each time the effect has been to extend rehabilitation services to increasing numbers and kinds of handicapped persons.

Originally, only the physically handicapped were included. Later the mentally ill and mentally retarded were brought in. Most recently – since 1965 – those who are vocationally handicapped in other ways, such as the alcoholic and the public offender, have been recognized and made eligible for rehabilitation services.

As the scope of the State-Federal program has broadened, its Federal funding base has expanded accordingly. Whereas originally the matching formula was a 50:50 one, Federal support has increased to the point that each State dollar appropriated for vocational rehabilitation earns $4 of Federal matching. (This matching ratio of 80:20 applies also to the vocational rehabilitation program for the blind and visually handicapped, and the ETR program for Public Assistance recipients.)

Funds are allocated to states on the basis of population and per capita income, but the allotment received is governed by how much the state itself appropriates to support its own basic program of vocational rehabilitation services.

Since the purpose of vocational rehabilitation is to provide a combination of services to persons whose handicaps interfere with acquiring or maintaining employment, a variety of services are authorized by both State and Federaal legislation. These include, but are not limited to:

— Comprehensive evaluation, including medical study and evaluation,
— Medical, surgical, and hospital care, and related therapy to remove or reduce disability,
— Prosthetic and orthotic devices,
— Psychological and psychiatric evaluation and therapy,
— Counseling and guidance services,
— Education and training services,
— Services in comprehensive or specialized rehabilitation facilities, including adjustment centers,
— Maintenance and transportation as appropriate during rehabilitation,
— Tools, equipment, and licenses for work on a job or in establishing a small business,
— Initial stocks and supplies,
— Placement services, including follow-up services to assist handicapped individual to secure and maintain their employment, and
— other goods and services necessary to render a handicapped person employable.

Any person is eligible for these services who (1) has a physical or mental disability which constitutes a handicap to employment, and (2) can reasonably be expected to benefit from rehabilitation services.

In general, most people are referred to the Division of Vocational Rehabilitation because (1) they need physical restoration, (2) they need help in emotional and personal adjustment, and (3) they need and want vocational

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counseling, vocational adjustment and/or training, and a job.

Referrals come to the agency from approximately 35 different sources, including the individual himself. Leading sources of referrals are public and private health and social welfare agencies, high schools, general hospitals, rehabilitation facilities, mental health centers, Social Security and State Employment offices, the Industrial Accident Commission, the applicant's employer or a family member, as well as former clients of the agency.

Of the approximately 2,500 new referrals to the agency during fiscal 1972, only 103 come from physicians.

The process of evaluation begins when first, the agency, and then the rehabilitation counselor to whom the case is referred, learn of the applicant as a prospect for services. The Counselor initiates a process of information-gathering which entails communicating with the referral source, interviewing the client, gathering medical information, school records, test records, etc. It becomes the counselor's function to determine, with the client and with the help of professional diagnosticians, what the client's individual circumstances are, and to work out with him a course of action to meet his or her needs.

During the initial phase of the evaluation, some persons are screened out who have been referred but are not eligible for service. Every effort is made to help them to obtain the particular service they are in need of.

On the other hand, this initial phase also serves to establish a tentative acceptance of a referral, and to suggest what additional information may be needed.

Specifically, this information will be of determining whether or not there exists a definable handicap which represents a serious barrier to employment; also, that there is reasonable expectation that rehabilitation services will enable this individual to become gainfully employed.

As a preliminary step in making such a determination, the counselor obtains a report of a recent complete medical examination. If the applicant has been hospitalized or examined within six months, the counselor requests a copy of his medical history. If a recent medical report is not available, the counselor arranges for the client to have a complete physical examination at VR expense.

Usually the client makes his own appointment and visits the physician of his choice. Customarily, he will have been furnished with a copy of the agency's General Medical Form and an Authorization and Invoice (BR-20) for professional services.

When the decision as to eligibility requires a specialist's evaluation, as in mental illness, emotional disturbance, mental retardation, loss of hearing, severe dental conditions, etc., this additional evaluation is arranged for as well. Special evaluation centers, hospitals, or other facilities may be utilized during this phase. If there is a question of the necessity for such a thorough-going evaluation, the opinion of one of the agency's district medical consultants is obtained.

After receiving information from the general medical examination, specialist's examination, or short-term stay in an evaluation facility, one of the following decisions is made:

— The case is moved into Extended Evaluation in order to obtain more information,
— Information received already precludes eligibility and the case is closed, after referring the applicant to another more appropriate agency,
— Information has been sufficient to establish eligibility and the counselor and client begin working out together a rehabilitation plan.

When it has been decided that further evaluation is needed before a client's eligibility can be determined, his case is moved into one of two extended evaluation classes. Most typically, a client undergoing evaluation at a facility, center, workshop, training site, etc. for a period exceeding 60 days would be placed in Extended Evaluation. The intent of an extended evaluation is to allow an extended period of time to evaluate the work potential of identified disabilities that are severe in nature before making a decision on eligibility or ineligibility.

Vocational rehabilitation services may be provided as necessary during a period not to exceed 18 months in the case of an individual whose disability is mental retardation; deafness; paraplegic; quadriplegic, or other spinal cord injuries or diseases; heart diseases; cancer; stroke; epilepsy; arthritis; mental illness; brain damage; cerebral palsy; muscular dystrophy; cystic fibrosis; or renal failure. The period of evaluation for other disabilities seldom exceeds six months.

When necessary to determine rehabilitation potential, the following services are proved in Extended Evaluation:

— Diagnostic and related services,
— Physical restoration,
— Maintenance,
— Transportation,
— Training and training materials,
— Interpreter services for the deaf,
— Comprehensive evaluation at rehabilitation facilities, workshops, etc.,
— Other services (e.g. job try-out, medication).

While in extended evaluation, clients are not provided job placement, occupational equipment, occupational licenses, or initial stocks.

Certification of an applicant's eligibility does not preclude doing further diagnostic evaluation to determine the specific services that the client might require or benefit from in being vocationally rehabilitated. The regular evaluation procedure, however –

— Identifies the physical or mental impairment,
— Defines functional limitations, explaining how they interfere with occupational performance,
— Establishes the need for VR services which will overcome or reduce these interferences, and
— Predicts the probable outcome of VR services.

Anyone who declines service, or has been processed through the referral and extended evaluation phases and been found *not* eligible for acceptance with the active caseload, is closed. Both the applicant and the referring

agency or individual are notified in writing of the action taken; an applicant who is dissatisfied with the decision may request a review and redetermination of his case.

Applicants who have been certified as eligible are designated "active cases," as such, they become active participants in their own rehabilitation, as they work together with the counselor, the physician and the other members of the rehabilitation team in drawing up a plan that will include one or more of the following services:

— Counseling and guidance,
— Physical restoration,
— Training, and
— Placement.

Counseling is an on-going process throughout the rehabilitation program of each individual client. For some, however, counseling, guidance, and placement are the only services required after evaluation to prepare them for employment. Clinical psychological counseling is (for VR purposes) termed psychotherapy and considered a "restoration" service. These are defined as "those medical and medically related services which are necessary to correct or substantially modify within a reasonable period of time a physical or mental condition which is stable or slowly progressive. They include:

— Medical or surgical treatment by general practitioners or medical specialists,
— Psychiatric treatment,
— Dentistry,
— Nursing services,
— Hospitalization, (either in-patient or out-patient care) and clinic services,
— Convalescent, nursing or rest home care,
— Drugs and supplies,
— Prosthetic devices essential to obtaining or retaining employment,
— Physical therapy,
— Occupational therapy,
— Medically directed speech or hearing therapy,
— Physical restoration in a rehabilitation facility,
— Treatment of medical complications and emergencies, either acute or chronic, which are associated with or arise out of the provision of physical restoration services and are inherent in the condition under treatment,
— Other medical or medically related services.

Before physical restoration services can be provided it must be determined (a) that the clinical status of the individual's condition is stable or slowly progressive (that is, the condition must not be acute or transitory or of so recent an origin that the resulting functional limitations and the extent to which such limitations affect occupational performance cannot be identified), and (b) physical restoration services must be expected to eliminate or substantially reduce the handicapping condition within a reasonable period of time. (As noted, they can also be provided to clients certified for extended evaluation.)

Personal adjustment, vocational adjustment, and vocational training services are key elements of the VR process. In fact, the provision of vocational training for handicapped persons as a service to help them overcome their vocational handicaps has been the real core of the program since its inception in 1921.

Although in recent years other aspects of the program have gained in prominence, vocational training and supplemental services such as maintenance, transportation, and training supplies still account for a major portion of the VR budget. With the increasing emphasis on higher skills for many jobs, it has been necessary for the Division to spend increasingly larger sums for this activity. Training may range from apprenticeship or on-the-job training through business or vocational school to a full four-year college program. The agency assumes, in whole or in part, the cost of tuition, books, fees, supplies, apprenticeship level tools and other equipment needed to support the client's training program.

Upon completion of the training period, the client is given help in finding suitable employment. However, it is understood by both client and counselor that the more effort the client makes on his own behalf, the better his chances of successful placement. It is at this time that the counseling relationship and goals are brought into sharp focus.

Once on the job, the client is given assurance that his counselor is still concerned with his progress, and may be called upon to give whatever assistance he can to facilitate his client's adjustment to the work situation. The first month on the job is considered by many to be the most critical point in the rehabilitation program. The counselor's judgment and sense of timing are extremely important in being able to lend support to the client while keeping him from developing any further dependency.

The basic criteria for terminating services to a client and reporting him as "employed" are:

— The program of rehabilitation services has been completed insofar as possible,
— Substantial rehabilitation services have been rendered the client,
— The client is suitably employed.

Cases closed as rehabilitated must as a minimum have –

— been declared eligible,
— received appropriate diagnostic and related services,
— had a plan of VR services formulated,
— completed the plan as far as possible,
— been provided counseling and one or more *other* rehabilitation services,
— been in Employed Status for a minimum of 30 days,
— indicated by substantial evidence, before closure, that the client is suitably employed in accordance with his needs and limitations.

During fiscal 1972, the Division served a total of 5,330 applicants and active cases, more than 3,000 of whom were carried over from the previous year. This represented an increase of 430 cases over the number served

in fiscal '71. Of the 2,681 in active status, 726 were closed as successfully rehabilitated in employment. Persons closed after referral or extended evaluation numbered 1,194. Those in referral status at the end of the year accounted for 1,254 of the total, and an additional 200 clients were still in extended evaluation.

During the year, more than 700 persons were provided with some sort of training, and more than 600 received physical restoration services.

Based on estimates submitted by counselors in the field, the Division expects to receive nearly 3,000 referrals in fiscal '73 and accept more than half of them. Of these new cases, together with those carried over from last year, the Division expects to close 900 persons in employment, or nearly 200 more than were closed rehabilitation in fiscal '72.

The Division, which has its central office in the Bureau of Rehabilitation, 32 Winthrop Street, Augusta, also maintains district offices in Bangor, Lewiston, Machias, Portland, Presque Isle, Rockland and Sanford. Each one is located in the same building as other Bureau of Rehabilitation and Bureau of Social Welfare field offices serving the same regional areas.

In addition to field services, the Division has been able to implement a number of special prorgams such as the cooperative VR units at each of the State's correctional institutions, a special unit at Biddeford High School to provide services primarily to the mentally retarded but also to other physically and mentally handicapped, and a cooperative VR unit at the Augusta State Hospital to serve both inpatients and outpatients.

Other services available are those of a Bureau staff specialist in the Disability Determination Division whose duties are primarily in the area of counseling with Social Security disabilities clients, and a Facilities Specialist whose consultative services are available to organizations, groups or individuals in finding out about federally funded grants available through Vocational Rehabilitation in Maine.

During the past ten years, the agency has provided approximately $2,000,000 in various types of grants-in-aid, with various kinds of matching ratios, to a number of rehabilitation facilities and programs throughout the State. Among them have been facilities, that provide work evaluations and work adjustment training for the handicapped; speech and hearing services, and two halfway houses for alcoholics. In addition, it has supplied special technical assistance to various agencies, including technical equipment for evaluation purposes.

In proportion to the extent of need, however, the nature and kinds of special assistance programs to the handicapped that the agency has been able to engage in and help or implement has been greatly curtailed by lack of funds.

As noted earlier, Maine's vocational rehabilitation program is a state-federal partnership in which the Federal Government participates on a 4 to 1 ratio. Over the years, the Maine Legislature has consistently fallen short of appropriating sufficient funds to earn anything like Maine's full federal allotment.

For this fiscal '73, for example, Maine was entitled, on the basis of its population and per capita income, to $4,200,000 in federal funds for its VR program. The 105th Legislature, however, appropriated only enough to match 35% of that grant-in-aid. The unearned balance was distributed among states that put forth the effort to meet with full matching potential.

The number of handicapped persons served by the Division last year (fiscal '72) represent 14% of the estimated 35,000 Maine citizens whose physical and mental impairments are of such severity that they are clearly within the criteria of disability, vocational handicap, and feasibility and thus are entitled to vocational rehabilitation services.

When the extent of this unmet need is considered alongside the social and economic benefits of enabling handicapped persons to become taxpayers instead of tax consumers, the challenge this presents to all of us, both as service providers and as the taxpayers comes into clearer focus.

Dates For Your 1973 Calendar . . .

Interim Meeting, M.M.A. House of Delegates — April 8

M.M.A. Annual Session — June 16-19

NEW EDUCATIONAL BOOKLET AVAILABLE

Blue Shield, as a part of its continuing public education program on drug abuse, is aiming at a new age group with a children's book entitled, "Happy Valley and the Drug Witch."

Since 1968, Blue Shield has been conducting a community oriented educational program with emphasis mainly on bringing facts regarding drug abuse to both young and older adults. Now, however, the emphasis is on "pre-readers" in the age group, 4-8.

According to Ned F. Parish, president of the National Association of Blue Shield Plans, "Drug abuse constitutes one of our most serious community problems, and clearly more must be done about it.

"Unfortunately, the very young have been largely neglected in drug abuse education," he said, "but we believe this new booklet and film will be good attitude conditioning tools."

Blue Shield has produced a six-minute animated film based on the booklet which will be offered to television stations, and subsequently to schools, kindergartens, churches, day camps, etc.

The booklet is not designed to teach the children facts about drug abuse, but rather its purpose is to condition their attitudes to the fact that "dope is bad for boys and girls." Parish points out that more sophisticated educational materials are made available to students as they reach advanced levels in school.

"Happy Valley and the Drug Witch" is a fable about an evil witch who turns herself into a pretty young girl and goes around trying to persuade other children to try her dope that "tastes just like candy." When they do, the girls and boys are turned into ladybugs and beetles, and the drug witch puts them in a cage in her house located in the deep forest surrounding Happy Valley.

The only way she can be stopped is if some child will refuse the evil dope, which of course, eventually happens. At that point, the pretty young girl turns back into the ugly witch and then into a wisp of smoke which is blown away by "the good wind which blows no evil." All the ladybugs and beetles are turned back into girls and boys, and happiness returns to Happy Valley.

Parish, in announcing the new additions to the Blue Shield program, points out that every parent and teacher with small children should read the book to his or her youngsters.

"If we are to make substantial progress in the fight against drug abuse, we must concentrate on the very young. Drugs are not something the child should first hear about on the street. The home and classroom are more appropriate environments for the training process," the NABSP president stated.

Both the booklet and film will be available through the Public Relations Department of local Blue Shield Plans. Other booklets available include, "Drug Abuse," "The Chemical Cop-Out," and "The Alcoholic American."

Since its inception in 1968, the public information program begun by Blue Shield has included five films which were presented as television specials, and three booklets.

The Drug Abuse and Alcoholic American booklets are available in quantities of 100 or less at no cost to Maine schools, civic groups, and concerned individuals. The accompanying films are also available at no cost and can be obtained by writing to the Public Relations Department, Maine Blue Cross and Blue Shield, Portland, Maine 04101, or by calling 775-3536, ext. 203.

Necrologies

Frederick J. Pritham, M.D.

1880-1972

Dr. Frederick J. Pritham, 92, of Greenville Junction, Maine's most famous country doctor, whose practice carried him through 5,000 square miles of Northern Maine wilderness, died on November 9th.

He was born in Freeport, Maine on July 6, 1880, son of Charles and Eleanor Merrill Pritham. Dr. Pritham was graduated from Freeport High School in 1900, received his medical degree from Bowdoin Medical School in 1905 and located in Greenville Junction where he practiced medicine for 67 years.

In a 1971 biography, "The Big Little World of Doc Pritham," Dorothy Clarke Wilson quoted Pritham as saying: "It's a doctor's business to go where he's called, come hell – yes, or fire, rain, snow, mud, fog, wind, ice – as well as high water!"

Small and wiry, "Doc" once figured that he logged at least 1,000 miles walking through the backwoods to care for the sick. Even as a student, he learned the rigors of long walks, commuting on foot several times a week from Freeport to Bowdoin College in Brunswick, nine miles away.

During the influenza epidemic of 1918, he treated more than 300 cases of flu and 10 of pneumonia, managing to catch brief moments of rest whenever he could – on his boat, in a crude lumber camp bunk or on the ground, wrapped in a blanket.

Finally, citizens of Greenville held a meeting and unanimously agreed that Dr. Pritham must abandon his tasks long enough to obtain at least three hours of sleep in each 24.

Dr. Pritham's fondest hobby was music, and he took pride in his honorary membership in the Greenville High School Band. A familiar figure at rehearsals and performances, he was always on hand to blow his E-flat alto horn to celebrate a Greenville High basketball victory.

Dr. Pritham was an honorary member of the Piscataquis County Medical Society and the Maine Medical Association, receiving a 50-year pin in 1955, 55-year pin in 1960, 60-year pin in 1965 and 65-year pin in 1970. He also served as doctor for the Canadian-Pacific Railroad for 50 years, was president of the Piscataquis County TB Association, a member of the State TB Association and was honorary chairman of the Christmas Seal drive in 1971.

Surviving are his widow, the former Sara Ring of Greenville Junction; one son, Dr. Howard C. Pritham of the Canal Zone; and seven grandchildren.

Aaron Cook, M.D.

1909-1972

Word has been received that Dr. Aaron Cook died in the late fall.

Dr. Cook was born in the year 1909, and received his medical degree from Jefferson Medical College in 1934.

He was a member of the Kennebec County Medical Association, the Maine Medical Association and the American Medical Association.

Donald E. Troop, M.D.

1929-1972

Dr. Donald E. Troop, 43, of Springvale, Maine, died on September 6. He was born in Boston, Massachusetts on August 15, 1929, son of Hobart Wellington and Rose Gillespie Troop.

He attended the University of New Hampshire from 1946 to 1948 and 1953 to 1954. Following six years of active duty with the U.S. Navy, Dr. Troop was graduated from Wayne University in 1955 and received his medical degree from the University of Michigan Medical School in 1959. He interned at the St. Benedict's Hospital in Ogden, Utah and then practiced in Ajo, Arizona from 1960 to 1968 before coming to Springvale.

Dr. Troop was a member of the York County Medical Society, the Maine Medical Association and the American Medical Association.

Oscar W. Perrault, M.D.

1903-1972

Dr. Oscar W. Perrault, 69, of Biddeford, Maine died at his home, October 16, following a brief illness.

He was born in St. Ferdinand, Canada on April 1, 1903, son of Simeon and Adeline Larose Perrault.

Dr. Perrault was graduated from St. Andre's School, Montreal College and received his medical degree from the University of Montreal Faculty of Medicine in 1932. He took postgraduate courses at the Cook County Hospital in Chicago and the Philadelphia Lying-In Hospital.

A practicing physician and surgeon in Biddeford since 1932, Dr. Perrault was a member of the York County Medical Society, the Maine Medical Association and the American Medical Association. He also served as First President of the Notre Dame Hospital in Biddeford, was a past secretary of the Notre Dame Hospital, served as Biddeford City Physician, City Health Officer and was awarded the congressional selective service medal for "faithful and loyal service" during World War II.

Surviving are his widow, Dorothy Seidel Perrault; a son, John of Rye, New Hampshire; a daughter, Mrs. Thomas Wallace of Cohasset, Massachusetts; a niece, Mrs. Arnold Cote of Biddeford; two sisters, the Misses Anna and Rose Perrault, both of Biddeford; two brothers, Urbain of Fort Lauderdale, Florida and Donat of Ontario, Canada; two grandchildren and several other nieces and nephews.

Francis M. Dooley, M.D.

1895-1972

Dr. Francis M. Dooley, 77, a general practitioner in Portland, Maine for nearly half a century, and former chief of staff and chief of obstetrical services at Mercy Hospital, died on November 22 after a long illness.

He was born in Portland on July 20, 1895, son of John H. and Hannah Cassidy Dooley. Dr. Dooley was graduated from Portland High School, Holy Cross, attended Bowdoin Medical School and received his medical degree from Yale Medical School in 1922.

Dr. Dooley was an honorary member of the Cumberland County Medical Society and the Maine Medical Association, receiving a 50-year pin in 1972. He was also a member of the American Medical Association and a past president of the American Academy of Family Physicians. In 1959, Dr. Dooley was made a Knight of the Order of St. Gregory the Great by Pope John XXIII.

Surviving are his widow, the former Evelyn Richard; a daughter, Miss Frances Dooley of Portland; a son, Dr. John R. Dooley, with the Armed Forces Institute of Pathology at Walter Reed Hospital; a sister, Miss Catherine E. Dooley of Portland; two grandchildren and several cousins.

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The Demonstrated Effectiveness of Cardiopulmonary Resuscitation

A Three-Year Experience in a Community Hospital Without House Staff

An Outcome Study

RICHARD T. CHAMBERLIN, M.D.*

INTRODUCTION

There are few physicians, perhaps there are none, who question the historical and theoretical aspects of cardiopulmonary resuscitation. The work in the laboratory has been quite convincing. It has shown that it is possible to produce an effective cardiac output by properly applied external chest compression and that fatal arrythmias may be corrected by electric countershock. It has also been shown that an adequate pO_2 can be produced by mouth to mouth breathing and that this is improved upon by using various assistive devices such as endotracheal tubes and/or mask and bag attachments.[1]

Questions have been raised, however, when attempts are made to relate cardiopulmonary resuscitative efforts to the entire medical community taking into account such frequently unknown variables as the time between the onset of the "episode" and the recognition of the event, the time between the recognition of the event and the application of proper resuscitative techniques, and the degree of accuracy of the diagnosis of the pathologic and pathophysiologic processes causing the "episode." This is particularly true in hospitals which have no physician house staff or other full-time salaried physicians who are going to be on the premises twenty-four hours a day to conduct optimum resuscitative efforts.

Many articles have been written documenting the clinical effectiveness of cardiopulmonary resuscitation both in immediate and in long-term survival. The largest number of reports come from university hospitals.[2–9] There have also been reports of similar experience from community hospitals.[10–13] The setting of the community hospital needs further definition, however, since in one of the above reports the hospital has six hundred and fifty-four beds[12–13] and in another there are five hundred and fifty beds and in each there is a staff which includes house officers. In Maine many hospitals consider themselves to be community hospitals yet they differ in many parameters from other, often larger and better staffed institutions that use the same designation. It may be important to draw this distinction when conducting studies of medical care in the different settings.

This paper summarizes a study of the results of establishing an effective program of cardiopulmonary resuscitation in a small community hospital which has no house staff. It is not an exhaustive study. Since refinements in the procedures and training programs involved were being made constantly throughout the three years covered in the report, it is difficult to attempt to make comparisons at any point in time with any previously adopted standards and criteria. The method of reporting cases and recording data about them also was imperfect resulting in an unfortunately large number of cases for which complete data is missing. Recognizing these defects, it is felt that it is important to document the effectiveness of medical care in all medical settings, large or small, hence the report. The report is also made to demonstrate a method of evaluating the quality of medical care which is often not well described today – an outcome study. Most studies concentrate on the measurement of processes of rendering of medical care and do not emphasize greatly enough the eventual outcome of such care. The present report attempts to use the method in the same context that it has been used in similar reports documented in the bibliography.[9,10,11,13]

*Thayer Hospital, Waterville, Maine 04901.

Description of Hospital, Staff and Training Programs

The study was accomplished in the years 1970-1972 at the Thayer Hospital, a community hospital of one hundred and seventy-four beds in Waterville, Maine. The hospital and its active staff of approximately fifty physicians serves a geographic area in the central part of Maine having a population of approximately 130,000 people. There are two other community hospitals in the same city and the most immediate service area is served by an ambulance service which is based at one of the other hospitals. Thayer Hospital has an emergency room staffed by nurses and covered by the physicians on the staff on a rotating basis. There is no requirement that the physician be physically present in the emergency room twenty-four hours a day. The emergency room is equipped with a cardiac monitor defibrillator; the ambulance service is not. The ambulance and the hospitals are linked by an areawide two-way communication network with a central dispatcher at the local police station. The hospital also has both an intensive care unit and a coronary care unit which are equipped with cardiac monitors and defibrillators and there is telemetry capability within the hospital. The coronary care unit was opened just shortly after the onset of the study period and its own organization, staffing and training programs were developing during the period covered by the study.

In 1968 an extensive in-service educational program was directed towards the problem of recognizing and treating cardiac arrest in the hospital. The program included physicians, nurses at all levels, nurses aides and orderlies. It followed the suggested guidelines of the American Heart Association.[14–15] Although the results of such training programs were monitored by the Patient Care Committee, the problem was considered important enough that a separate committee called the Code 99 Committee was established in 1970. This committee has met monthly for the following purposes: (1) To establish, review and modify policies and procedures for the conduct of emergency resuscitative measures throughout the hospital. (2) To review and recommend to the Executive Committee of the Medical Staff and to the Hospital Administration the purchase of equipment for such resuscitative efforts. (3) To monitor the results of the resuscitation efforts and use this information as a basis for further in-service educational programs. It is as a result of the latter function that this study was accomplished.

Although it is important to document all of the above activities of the Code 99 Committee, this report will not detail the steps in the Code 99 protocol nor the equipment selected, but rather will describe the third function of the committee – the monitoring of the results of the entire effort.

Definitions

An emergency event is defined as any event which in the opinion of the discoverer or observer is life threatening and for which immediate help is needed. The term is used to identify such events is Code 99. These are usually events which are sudden and unexpected in their origin, and although the hospital personnel have had a training program as described above, no attempt was made to adhere to a strict set of criteria designed specifically for cardiac arrest. This differs significantly with reports of some authors in which criteria such as no audible heart sounds, no recordable blood pressure, respirations or pulse, dilated pupils, and pallor with or without cyanosis were used.[11]

A short-term survival is defined as recovery from the emergency event itself by re-establishment of a clinical condition which although in some instances may be considered precarious, nevertheless represents a reversal of the immediate life threatening situation.

A long-term survival is defined as recovery from the event to allow discharge from the hospital at some later date.

Method of Study

The Code 99 protocol directs that data shall be gathered at the time of an emergency event by one of the nurses in attendance. This data is recorded on a form, the design of which has been studied and revised by the Code 99 Committee. The data on the completed form is then coded and transferred to the McBee Keydex System to allow for storage and future retrieval for study. The results of these studies serve as the bases for the in-service educational programs as well as to identify equipment and/or personnel needs.

One of the difficulties in the study was that of incomplete reporting of the data. This has resulted in a situation in which some of the following report is well substantiated by complete data and some of which is not as complete. The display of incidence data covers a seven-year period. Follow-up and result data is available for only three years and is the main theme of the report.

Table 1

Year	Total Deaths	Attempted Resuscitations	Percentage
1966	125	27	21.6%
1967	142	23	16.1%
1968	139	25	17.9%
1969	161	26	16.1%
Sub Total	567	101	17.8%
1970*	173	32	18.4%
1971	200	40	20.0%
1972(10 mo)	197	44	22.2%
Sub Total	570	116	20.2%

*Prior to and including 1970 patients brought to the emergency room apparently dead on arrival were not recorded as admissions and, therefore, do not appear in total deaths. From 1971 on they are so recorded. There were eleven cardiopulmonary resuscitation attempts in the emergency room that year so that the data prior to 1970 is not totally comparable to that of 1971 and 1972.

TABLE 2

YEAR (TOTAL)	TIME OF DAY 7 AM-3 PM	3 PM-11 PM	11 PM-7 AM	LOCATION ER	ICU	CCU	MED SURG	PEDI OBS	X-RAY	OTHER
1966 (27)	12	14	1	9	11		5		2	
1967 (23)	10	12	1	7	8		7		1	
1968[a] (25)	8	4		5	6		13		1	
1969[a] (26)	16	7	1	8	7		10			EEG-(1)
1970 (32)	13	16	3	10	7		10	1	1	PT-(1) OR-(1) BUS Office-(1)
1971 (40)	21	13	6	12	5	5	15	[b]	2	
1972 (44)	14	23	7	11	3	22	7		1	
TOTALS (Percent)	94 (46.7%)	89 (43.7%)	19 (9.4%)	62 (28.7%)	47 (34.2%)	27	67 (31.0%)	1	8	4

(a) In 1968 thirteen reports did not specify AM or PM. In 1969 there were two such reports.
(b) Obstetric Service was transferred to another hospital in 1971.

RESULTS AND ANALYSES

Incidence of cardiopulmonary resuscitation attempts. The incidence of attempts at cardiopulmonary resuscitation was studied in relation to the total number of deaths, excluding stillbirths and newborn deaths, which occurred in the same reporting period. The incidence is also shown for several years prior to the 1970-1972 reporting period in an attempt to study the effect the in-service education programs of 1968 and 1969 may have had on the number of emergency situations recognized.

These results compare favorably with those of Lemire, et al, who reported an average of 20.2% attempts related to total deaths in their ten-year study with a range of 7.1% to 28.8%.[9]

There is only a slight increase in percentage attempts in the present series since the in-service programs and the deliberations of the Code 99 Committee. The author has been unable to find any note in the literature of what might be an expected incidence in a hospital of this size.

Location and time of emergencies. Table 2 displays the distribution of emergency events by the time of day and by nursing unit involved. Although the hospital has a larger number of nursing units than indicated in the table, certain common types of units are combined for the sake of brevity. The data has some definite inaccuracies: (A) Completeness of reporting varied both by time of day and by the unit involved. (B) The Coronary Care Unit was opened in January 1971. Initially the CCU protocol directed that no Code 99 would be necessary in CCU because the equipment and level of nurse training in the unit would make it unnecessary. In 1972 it was decided that if a nurse in CCU determined that she needed assistance she might have the option of initiating a Code 99. Thus, the table shows the CCU experience in 1972 as quite different from the previous year.

There are no comparable data in the reports studied and listed in the bibliography nor would one expect them. This analysis did help the Code 99 Committee in identifying areas in the hospital in which to concentrate in-service training programs.

Outcome Studies

A. Completeness of reporting. At the time the Code 99 Committee first attempted to study the outcome of the Code 99 procedure, it became apparent that incomplete reporting would be a problem. Although incidence data reported above was available since 1966, there was no mechanism available for easy retrieval of records of patients who had a Code 99 event. A reporting system was instituted in late 1969 and the remainder of the results will be limited to the years 1970-1972. Even after the reporting system was in effect, incomplete reporting reduced the number of reports which were suitable for study. This is summarized in Table 3.

TABLE 3

YEAR	TOTAL CODE 99'S CALLED	USABLE REPORTS	% USABLE
1970	32	16	50%
1971	40	28	70%
1972	44	34	77%
Total	116	78	66%

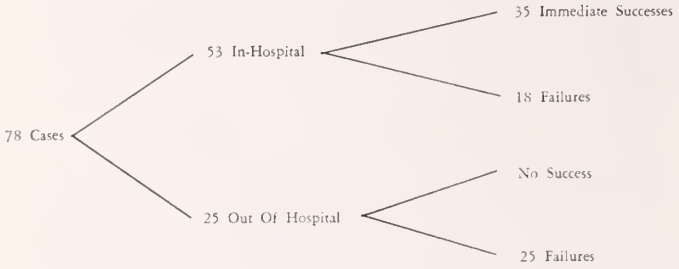
For the benefit of the in-service educational needs of the hospital, the incomplete reports were analyzed as to ward and time of day in order to institute corrective measures aimed at securing as close to 100% reporting as possible.

The remainder of the report is based on the seventy-eight usable reports.

B. Immediate success as related to the location of the emergency. From the very outset of the study, it was apparent that there were virtually no survivors among those patients who were brought to the emergency room by ambulance. The exact circumstances in such cases are always difficult to elucidate. Frequently the ambulance personnel have felt that in their judgment a patient was living when the ambulance arrived and emergency cardiopulmonary resuscitation techniques were applied at the scene and in the ambulance during transportation. In most of these instances, the ambulance personnel radioed the Code 99 signal to the hospital while enroute. From 1971 on, all of these patients had the Code 99 protocol continued upon arrival at the hospital emergency room and were officially entered as admissions to the hospital for statistical purposes.

Table 4 shows the total lack of any success, even immediate success in the group described above.

TABLE 4



It is also noteworthy that of those emergencies which occurred in the hospital there was a 66% immediate success rate.

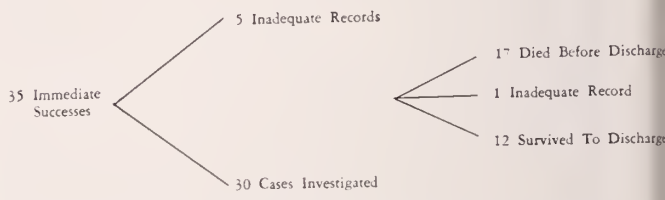
The immediate success rate varies by location within the hospital and by time of day as shown in Table 5. Note should be made that there are two separate groups of patients reported in the emergency room. The first group described above are brought in by ambulance. The second group are those already in the emergency room and who have an emergency occur while there.

TABLE 5

LOCATION	IMMEDIATE SUCCESS	FAILURE
Emergency Room		
1. Outside Case	0	25
2. Inside Case	2	2
ICU	2	6
CCU	18	4
Medical-Surgical	9	5
Pediatric-Obstetrical	1	0
X-Ray	1	1
Other	2	0
Total	35	43
SHIFT		
7AM-3PM	13	23
3PM-11PM	14	15
11PM-7AM	8	5

C. Long-Term Survivals. Next, an attempt was made to follow-up the thirty-five immediate success cases. Again, the study was hampered by incomplete and/or inadequate records.

TABLE 6



Of those who survived the episode but expired before discharge the actual length of survival is shown as follows.

TABLE 7

	DAYS OF SURVIVAL FOLLOWING CARDIOPULMONARY RESUSCITATION						
	1	2	3	4	5	6	7
Number Of Cases	6	3	4	2	1	0	1

Table 8 compares the long-term survival rate in this study with some of the experience documented in the literature.

TABLE 8

STUDY	LENGTH OF STUDY	TOTAL # OF PTS.	# LONG-TERM SURVIVAL	% LONG TERM
Present Study	34 mos	78	12	15.3%
Hofkin[16]	9 mos	78	9	11.5%
Lemire[9]	10 yrs	1,204	230	19.1%
Medford[10]	4½ yrs	56	10	17.9%
Sandoval[11]	6 mos	26	8	30.7%
Jersaty[13]	3 yrs	237	52	22.0%

DISCUSSION

Allowing for differences in all of the basic variables such as definition of terms and methods of reporting, it is of interest that a relatively small community hospital without house staff can perform favorably compared to larger teaching centers in the area of cardiopulmonary resuscitation. This should not be surprising if one recalls the basic knowledge on which a cardiopulmonary resuscitation effort sets. The only tool one really needs is a knowledge of the pathophysiology such that immediate and accurate mouth to mouth breathing and chest compression begins as soon as the emergency is identified. The most crucial comparative data is not available in this study. That is, what was the immediate and long-term survival rate prior to 1969 when the complete cardiopulmonary resuscitation training program was implemented? In any event, the study would seem to lend added weight to the arguments that cardiopulmonary

resuscitation efforts are worthwhile and that all hospitals, large or small, should have a well-rehearsed procedure ready to respond to such emergencies.

The study also clearly defines a large area of future educational need – the community at large – in the service area of this hospital. Recent composite data from both rural and urban areas have shown an immediate survival rate of 60.7% in cases receiving cardiopulmonary resuscitation by trained ambulance personnel outside of and prior to reaching the hospital.[17] At present a mechanism is being developed to study this aspect of the problem in the geographic area covered by this report.

In spite of these problems, all of the reports noted here as well as the present one attempts to analyze a specific method of medical care by the outcome of such care. Since the actual process of medical care rendered in the area of cardiopulmonary resuscitation does not vary significantly across the nation, variations of outcome must be related to such input variables as the size and type of hospital, presence of trained personnel, equipment available, etc.

One often hears the excuse given that the medical care rendered is as it is because the hospital is too rural, too small, or too understaffed. The courts, on the other hand, have moved away from purely local definitions of quality care and national and/or regional standards have been applied. Thus, it behooves the medical profession to adopt a continuing program of study of the quality of medical care rendered not only by the process of such care but also by the outcome of such care.

Summary

A thirty-four month study of the effectiveness of cardiopulmonary resuscitation in a 170-bed community hospital which does not have a full-time house staff is reported. The incidence of cardiopulmonary resuscitation efforts (20.2% of all deaths), the immediate survival rate (66.0% of 78 cases studied), and the long-term survival rate (15.3% of the 78 cases studied) compare favorably with reports from larger, well staffed institutions. Additional data of interest to local in-service education programs is also shown. The study represents an example of an attempt to document the quality of medical care based on the outcome of such care.

This report also represents an attempt to approach an even more complex issue – the study of medical care itself. This is complex because of the large number of variables involved whenever man confronts his environment. No study can be called technically accurate unless all variables are well defined and are measured.

References

1. Stephenson, Hugh E., Jr.: Cardiac Arrest and Resuscitation, C. V. Mosby Co., St. Louis, 1969, pp. 3-40.
2. Jude, J. R., Kouwenhaven, W. B., and Knickerbocker, G.: External Cardiac Resuscitation, Monograph, Surgical Science, 1:59-117, 1964.
3. Grace, W. J., and Minoque, W. F.: Resuscitation For Cardiac Arrest Due To Myocardial Infarction, Diseases of the Chest, 50: 173-175 (Aug), 1966.
4. Klassen, G. A., et al: Cardiac Resuscitation in 126 Medical Patients Using External Cardiac Massage, Lancet, 1: 1290-1292, 1963.
5. Lillehei, C. W., et al: Four Year's Experience With External Cardiac Resuscitation, J.A.M.A., 193: 651-658, 1965.
6. Johnson, A. L., Tanser, P. H., Ulan, R. A., et al: Results of Cardiac Resuscitation in 552 Patients, American Journal of Cardiology, 20: 831-835, 1967.
7. Baringer, J. R., et al: External Cardiac Massage, New England Journal of Medicine, 265: 62-65, 1961.
8. Himmelbach, S. R., et al: Closed-Chest Cardiac Resuscitation, New England Journal of Medicine, 270: 118-122, 1964.
9. Lemire, J. G., and Johnson, A. L.: Is Cardiac Resuscitation Worthwhile, The New England Journal of Medicine, 286: 970-972, 1972.
10. Medford, F. E.: Cardiac Arrest In The Community Hospital, West Virginia Medical Journal, 62: 43-46, 1966.
11. Sandoval, R. G.: Survival Rate After Cardiac Arrest In A Community Hospital, J.A.M.A., 194: 675-677, 1965.
12. Jeresaty, R. M., et al: Experience With External Cardiac Resuscitation In A Community Hospital, Connecticut Medicine, 32: 193, 1968.
13. Jeresaty, R. M., Godar, T. J., and Liss, J. P.: External Cardiac Resuscitation In A Community Hospital, Archives of Internal Medicine, 124: 588-592, 1969.
14. Jude, J. R., Scherlis, L., and Farr, M. E.: Emergency Measures In Cardiopulmonary Resuscitation, Discussion Guide, American Heart Association, 1964.
15. Dembo, D. H., Scherlis, L., and Judge, J. R.: Definitive Therapy In Cardiopulmonary Resuscitation, Discussion Guide, American Heart Association, 1965.
16. Hofkin, G. A., Cpt. MC, USAR: Survival After Cardiopulmonary Resuscitation, J.A.M.A., 202: 652-654, 1967.
17. Crampton, R. S., Aldrich, R. F., Stillerman, R., and Gascho, J. A.: Prehospital Cardiopulmonary Resuscitation In Acute Myocardial Infarction, New England Journal of Medicine, 286: 1320, 1972.

Dates For Your 1973 Calendar . . .

Interim Meeting, M.M.A. House of Delegates — April 8

M.M.A. Annual Session — June 16-19

Early Experience With Newer Agents in Bone Scanning

ANTHONY BETTS, M.D.*

In most breast cancer cases, it is the hope and motive of the surgeon to eradicate the tumor. This is the rationale for the radical mastectomy. Proof that the disease has spread beyond the confines of the planned operation is an indication for some lesser procedure or other mode of treatment.

The customary approach, therefore, has been to examine for local spread to nodes; to seek, by x-ray, evidence of lung metastasis, and by biopsy or scan spread of the tumor to the liver. It was only when bone pain or an obvious fracture presented itself that an x-ray examination of the skeleton has been made.

This was because methods for skeletal survey, until now, have been tedious, expensive, and exhausting to the patient. For years a search has been made for a quick and satisfactory isotope procedure and reliance was, until recently, placed upon the use of isotopes of Strontium. These all had limitations; its great affinity for the colon necessitated the examination to be spread across several days, and its high energy made handling difficult and results insensitive. Furthermore, the patient received a high dose of radiation. Thus, it was not practical for use in routine preoperative work-up of suspected cancer patients.

Within the last year research has rewarded the clinician with an almost ideal bone scanning agent in the form of Technetium 99m stannous polyphosphate.[1] The agent is excellent for bone scanning because it can be kept on hand at almost all community hospitals equipped with the usual nuclear diagnostic devices. The Technetium 99m can be eluted at will from the hospital generator by the usual milking procedure and added to a packaged amount of stannous polyphosphate and it is ready for immediate use. The patient may be injected and scanned within three hours. Because Technetium 99m is used, the gamma camera may be employed and the entire examination completed, in comfort, within an hour.[2] Clear Polaroid pictures are obtained, indicating regions of bone pathology.

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Our experience at the Thayer Hospital during the last two months has been limited to fifteen bone scans in cancer patients. Of these, six were clearly positive. From these six cases, four had initial metastatic x-ray series readings which were deemed negative for cancer. Two were subsequently proven to have metastatic bone tumor by biopsy. A further one was shown to be due to widespread osteoporosis of the vertebral column which had resulted in compression fractures. To date, there have been no false negative findings.

It is recognized that Technetium 99m stannous polyphosphate will give positive bone scan pictures in any bone pathology, be it due to fractures, Paget's disease, osteomyelitis, osteoporosis, etc. In most instances, however, in the cancer patient, these differential diagnoses may be ruled out by other means. The procedure has no known contraindications and the radiation exposure is minimal with bladder receiving the highest dose, that of 0.21 rads per MCi. The skeleton receives 0.07 rads per MCi. The short physical and biological half lives of Technetium polyphosphate means that no delaying procedures are required in the patient's care.

With this background and assuming that the results are acceptable, it would seem that the cancer surgeon could employ this simple method as a further means of staging lung, thyroid and renal cancer patients prior to any cutting procedure; and in particular, in breast cancer. In the latter instance a positive scan would preclude any radical surgery, the sole rationale of which is to prevent any further bodily spread.

However, of more subtle interest is the attitude of the oncologist who is now faced with the asymptomatic evidence of breast cancer spread to bone, revealed only by scan. His endocrine orchestration may well have to be modified in the immediate future of such cases.

REFERENCES

1. Subramanian, G., McAfee, J. G., O'Mara, R. E., et al: 99m Tc-Polyphosphate PP-46: A New Radiopharmaceutical for Skeletal Imaging. J. Nucl. Med. 12: 399-400 (1971).
2. Subramanian, G., McAfee, J. G., Bell, E. G., et al: 99m Tc-Labeled Polyphosphate as a Skeletal Imaging Agent. Radiology 102: 601-702 (1972).

Combined Scalp and Skull Resection for Basal Cell Cancer

Loring W. Pratt, M.D., F.A.C.S.,* Bruce Trembly, M.D., F.A.C.S.** and Irving Goodof, M.D., F.A.C.P.***

The criteria for adequate surgical treatment of cancer are satisfied only when complete three dimensional resection of a lesion with adequate margins is accomplished. Inability to adequately resect a malignant tumor results from a variety of circumstances, one of which is contiguity with or involvement of adjacent important structures.

In the head and neck, this problem is particularly significant, as here many important structures are found in close proximity, and the ability to make satisfactory three dimensional resections of tumors often results in sacrifice of such structures as well as in cosmetic deformity. In some instances, the importance of any particular structure or part of it is not as great as it might appear, and its removal, if thoughtfully done, may not significantly impair function of the patient.

Basal cell cancer is as a rule an easily treated neoplasm with relatively low potential for serious extension. When these tumors do become large and are recurrent, their natural history is different, and they are often aggressive, persistent lesions. Bone involvement is an ominous sign and connotes a malignant potential not ordinarily attributed to basal cell cancer. In more unusual instances, distant metastases are seen.

The literature contains several reports of cases in which inclusion of major portions of the skull were essential to adequate extirpation of a malignancy. Ketcham et al in 1963 utilized a combined intracranial and facial approach to cancer of the paranasal sinuses in nineteen cases. They felt that this approach permitted a true en bloc resection of cancer in an area which is difficult to approach surgically.

Conley, in 1965, reported on the application of temporal bone resection to the treatment of middle ear malignancy. Hanna, et al, in 1967, suggested a technique for temporal bone resection which was applied to cancer of the external auditory canal, the internal ear and the parotid gland with a five-year survival rate of 42%.

Terz, et al, in 1970, applied the principle of en bloc resection of the pterygoid region to the management of advanced oropharyngeal cancer in six cases. This approach involved partial craniectomy. Edgerton, et al, in 1965, proposed a combined intracranial-extracranial approach to resection of craniofacial malignancies and the use of a two-stage split flap technique for reconstruction. These reports are but a few of an extensive bibliography on this type of surgical approach. They do indicate the practicality of including an intracranial approach with partial craniectomy to provide adequate margins for the resection of a malignant craniofacial tumor.

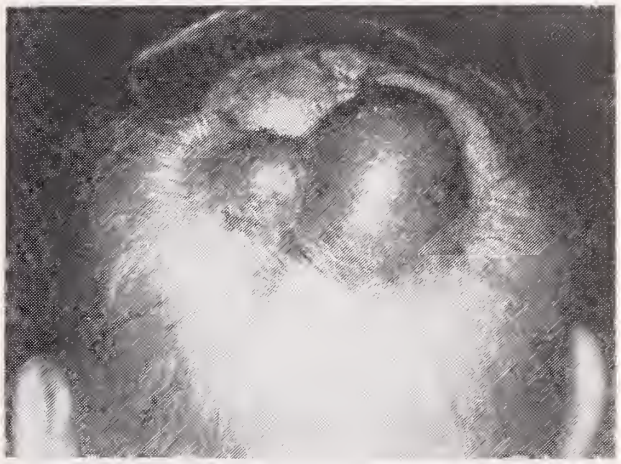

Fig. 1. The lesion as it originally presented, showing its thickened, cystic appearance with ridge of induration about the base. Lack of ulceration is especially to be noted.

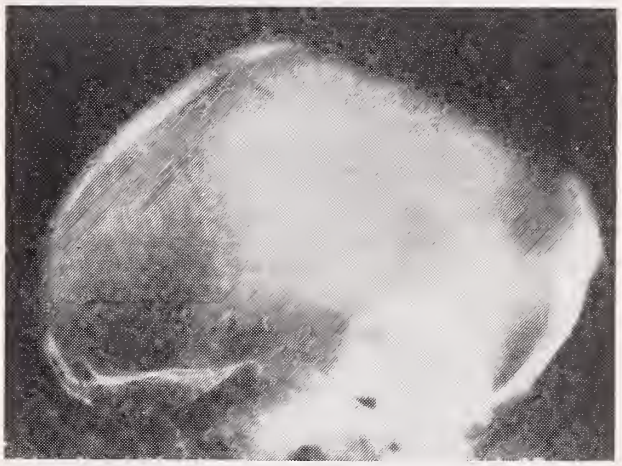

Fig. 2. Lateral x-ray of skull taken postoperatively demonstrating extensive area of bone removed at operation.

Case Report

An 85-year-old retired laborer was first seen in August 1967.

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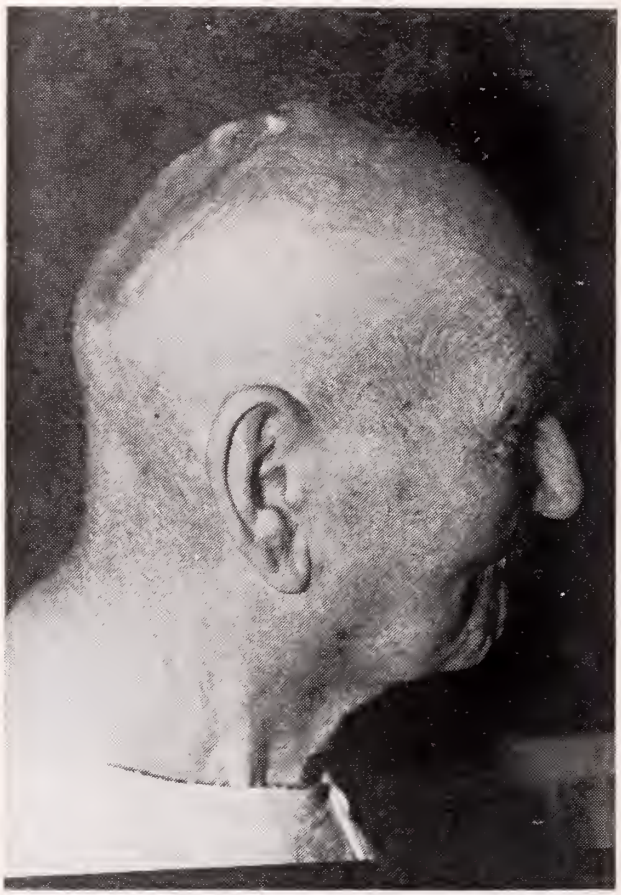

Fig. 3. Lateral view of patient showing large cranial defect covered with split thickness skin graft.

Fig. 4. Back of head protected by helmet designed to prevent damage to exposed area of brain.

He had been aware of a growth on his head for 3 to 4 years. The lesion was removed elsewhere in 1966, with a diagnosis of basal cell cancer, hair matrix type. The mass recurred in 3 months and he was told that he could have no further treatment for the scalp lesion. Two months prior to his initial examination he had also had a small mass removed from behind his left ear.

At his initial visit he had a 9x10 cm. lesion (Fig. 1), elevated but not ulcerated, which was fixed to his skull and surrounded by a thick ring of induration.

Skull x-rays disclosed an area of eburnation of the outer table of the skull in the parietal area just to the right of the midline.

Medical consultation impression: "Arteriosclerotic heart disease manifest by atrial fibrillation, mild congestive heart failure and possible recent myocardial infarction."

A resection of scalp together with a large flap of bone, extending across the mid-line, was carried out en bloc. There was no recognizable involvement of the inner table. A large split thickness skin graft took well and the patient was fitted to a helmet to protect the brain beneath the defect (Figs. 2, 3, 4, 5).

Pathology confirmed the origin of the tumor: "Basal cell carcinoma, sweat gland origin, invading bone (Figs. 6, 7, 8, 9)."

The patient made an uneventful recovery and was subsequently admitted in 1971 because of painless hematuria and underwent transurethral resection of a transitional cell carcinoma of the bladder with uneventful recovery.

He died in September 1971 at the age of 89 of a stroke, with no evidence of scalp tumor recurrence, regional or distant metastases. He lived four years following his surgery.

Discussion

Although adequate three dimensional resection of a malignancy of the head and neck may be impossible without the sacrifice of some major anatomical structure, this necessity need not preclude a successful surgical procedure.

Inclusion of a segment of skull in the tumor block by means of a combined intracranial-extracranial approach is a practical and useful concept in the management of extensive malignancy of the head and neck.

Conclusions

1. Three dimensional resection of tumor is necessary to provide adequate surgical extirpation.
2. Involvement of adjacent significant structures should not per se be considered indication of inoperability.
3. Segments of the skull may be included, en bloc, by combined intracranial and extracranial approach, with an adjacent malignancy in order to permit three dimensional resection of a tumor with adequate margins.

Acknowledgment

The authors wish to acknowledge the skillful help of Mr. Carl Nelson, Athletic Trainer, Colby College who made the prosthetic helmet-like device which was comfortably worn by the patient

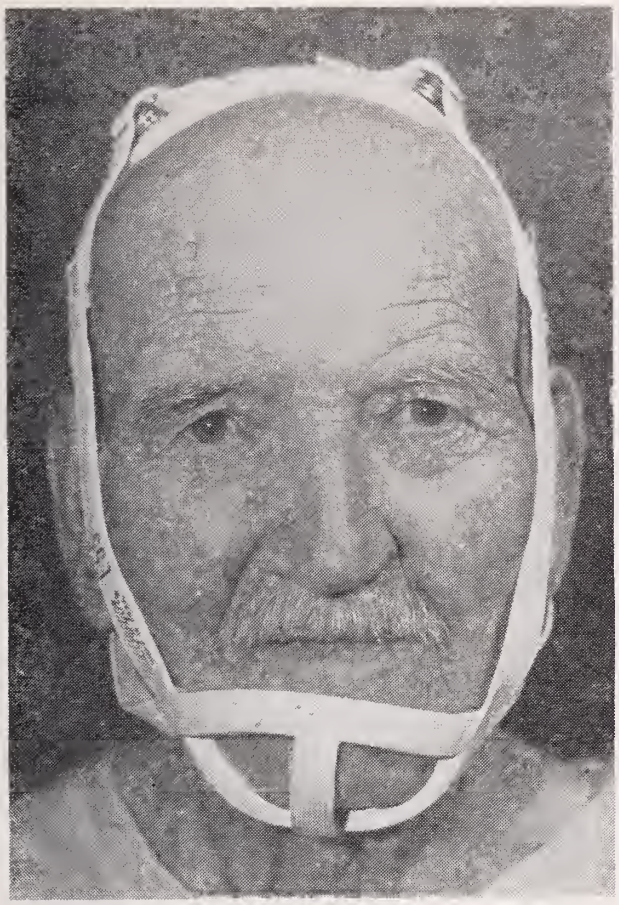

Fig. 5. Front view showing method of attaching helmet and keeping it in place.

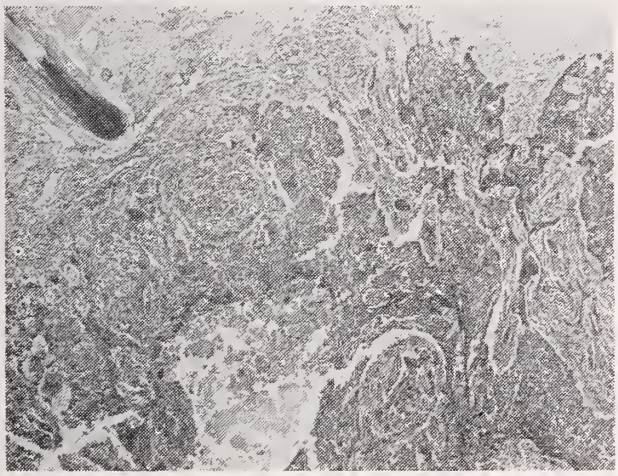

Fig. 6. Low power microscopic view of tumor of scalp showing typical basal cell cancer. (X50)

to protect him from injury throughout the remainder of his life.

REFERENCES

1. Ketcham, A. S., Wilkins, R. H., VanBowen, J. M., and Smith. R. R.: A combined intracranial facial approach to the paranasal sinuses. Am. J. Surg. 106, 698-703, Nov. 1963.

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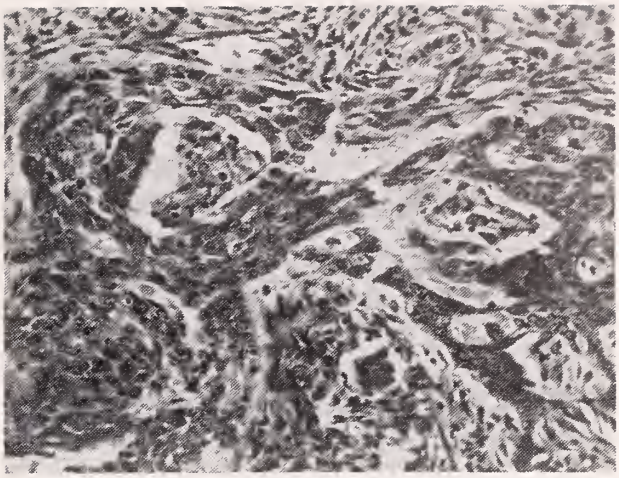

Fig. 7. High power microscopic view of tumor of scalp showing cellular detail of tumor. (X250)

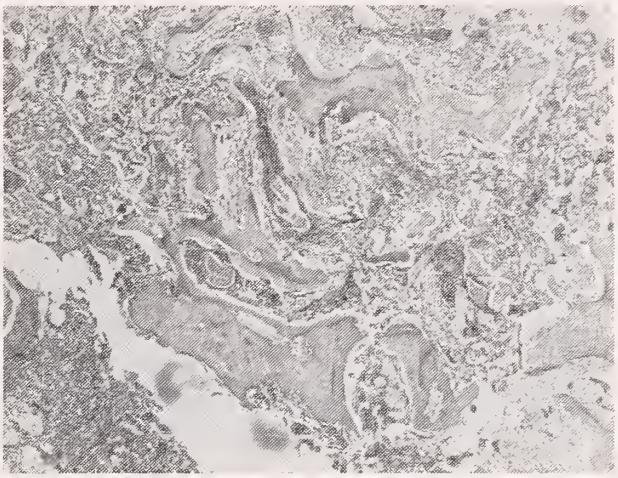

Fig. 8. Low power view of section of skull showing invasion of bone by tumor. This shows typical basal cell tumor invading the bone. (X50)

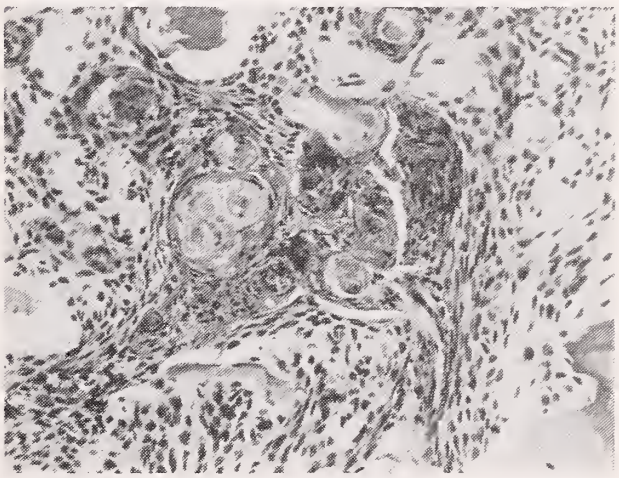

Fig. 9. High power view of bone of skull showing cellular detail of tumor invading the bone. In this view, keratinization is visible with formation of epithelial pearls. (X250)

Clinical Selection of Antibiotics

David R. Ginder, M.D., F.A.C.P.*

Clinical selection of antibiotics refers to the rational choice of antibiotics prior to isolation of microorganisms and performance of antibiotic susceptibility studies. Three kinds of information are required to make the correct early decision on antibiotics:

1. A reasonable working diagnosis.
2. Knowledge of the various microorganisms that may cause the clinical illness.
3. Awareness of the usual antibiotic susceptibilities of the most likely microbial causes of the illness under consideration.

These requirements are generally self-evident and well recognized with the possible exception of the third point. Tables 1, 2, and 3 illustrate aspects of the usual susceptibilities of common pathogenic microorganisms.

Microorganisms listed in Table 1 are always susceptible to the listed antibiotics. Therefore, the antibiotic of choice when these organisms are suspected as a cause of the clinical illness is quite evident. Selection of antibiotics for the microorganisms listed in Table 2 is usually straightforward but there are modifying factors which will be discussed. Even when organisms listed in Table 3 are suspected, there is a logical preference for certain antibiotics. Tables 1, 2, and 3 also indicate that only a small number of the available antibiotics need to be used in the treatment of infectious disease.

The practical aspects of making a rational choice of antibiotics are developed with a series of case presentations that illustrate common clinical problems.

Pneumonia

Mycoplasma pneumonia

Although mycoplasma pneumonia was a familiar and commonly diagnosed entity when it was known as primary atypical pneumonia, etiology unknown, there is tendency today to fail to consider it as a possible diagnosis. This is unfortunate because (1) it is easy to recognize in its classical presentation, (2) it is becoming common once again, and (3) it responds promptly to erythromycin or a tetracycline but not to penicillin.

Case 1. A previously healthy 38-year-old man was admitted acutely ill, with a six-day respiratory tract illness, characterized by a gradual onset of sore throat, bronchial congestion, and a nonproductive cough. Persistent headache was also a prominent symptom and was greatly aggravated by paroxyms of coughing. Fever was noted the second day of illness and gradually rose to 104°F by the day of admission. Rales were heard over the anterior aspect of the left chest. A dense infiltrate, confined to the anterior segment of the left upper lobe, was seen in the chest films. The leukocyte count was normal. Sputum was not discolored. Gram stain revealed a mixed gram positive and negative flora; few polymorphonuclear leukocytes were seen. On culture, viridans streptococci and Neisseria catarrhalis were the predominant organisms.

Table 1

Microorganisms With Constant Antibiotic Susceptibilities

Microorganisms	*Antibiotics* *First Choice*	*Alternative*
Groups A, B, C, and G streptococci	Penicillin G	An erythromycin
Diplococcus pneumoniae	Penicillin G	An erythromycin
Neisseria meningitis	Penicillin G	Chloramphenicol
Clostridium species	Penicillin G	A tetracycline
Haemophilus influenza	Penicillin G	Chloramphenicol
Pasteurella tularensis	Streptomycin	A tetracycline
Brucella species	A tetracycline	Chloramphenicol
Treponema pallidum	Penicillin G	A tetracycline
Rickettsia	Chloramphenicol	A tetracycline

Table 2

Organisms With Varying Antibiotic Susceptibility

Usual Antibiotic Susceptibility Provides Excellent Guidance in Selecting Antibiotics

Microorganism	*Antibiotics* *First Choice*	*Alternative*
Streptococci		
Group D	Penicillin G and streptomycin	Vancomycin
Anaerobic	Penicillin G	A tetracycline
Viridans	Penicillin G	Vancomycin
Shigella	Ampicillin	A polymyxin
Neisseria gonorrhoeae	Penicillin G	A tetracycline
Salmonella	Chloramphenicol	Ampicillin
Mycobacterium tuberculosis	INH and ethambutal	Rifampin
Bacteroides species		
Respiratory strains	Penicillin G	Chloramphenicol
Gastrointestinal strains	Chloramphenicol	Clindamycin

Table 3

Initial Treatment for Microorganisms Always Requiring Susceptibility Testing

Microorganism	*Antibiotics* *First Choice*	*Alternative*
Staphylococcus aureus	A penicillinase resistant penicillin	Vancomycin
E. coli	A polymyxin	Gantamicin
A. aerogenes	Gentamicin	Kanamycin
Klebsiella pneumoniae	Kanamycin	Gentamicin
Proteus mirabilis	Ampicillin	Gentamicin
Indole positive proteus	Kanamycin	Gentamicin
Pseudomonas species		
Urinary tract	A polymyxin	Gentamicin
Other infections	Gentamicin with carbenicillin	A polymyxin

*Formerly Professor of Medicine in Infectious Disease at the University of Missouri School of Medicine.

The patient was treated with erythromycin 500 mgs every six hours. Symptomatic improvement took place in 24 hours and the temperature was normal in 48 hours. The patient made an uneventful recovery. Cold agglutinins were present in a titer of 1:128 four weeks after the illness began.

Case 2. The 36-year-old wife of the patient described in Case 1 developed an upper and lower respiratory tract infections two weeks after her husband became ill. Her signs and symptoms were similar except fever was minimal despite a dense infiltrate in the posterior basilar segment of the right lower lobe. Other laboratory studies were within normal limits. Gram stain and culture of the sputum revealed normal flora. Symptomatic improvement occurred in 24 hours after erythromycin was begun.

COMMENT: These patients illustrate the familial incidence of mycoplasma pneumonia and the usual incubation period of about nine days to two weeks. The combination of headache, harassing paroxsyms of nonproductive cough, normal leukocyte count, normal bacterial flora in the sputum, and infiltrate in the lungs (including segmental and lobar distribution) should always suggest mycoplasma pneumonia.

Erythromycin is now regarded as the antibiotic of choice for mycoplasma pneumonia. Erythromycin is also the drug of choice when it is difficult to differentiate mycoplasma from pneumococcal pneumonia. However, when the differential diagnosis of pneumonia includes mycoplasma, psittacosis/ornithosis, Q fever and mild tularemia – a tetracycline is the drug of choice. Adenovirus infection may mimic mycoplasma pneumonia but it does not respond to erythromycin or tetracycline.

Pneumococcal pneumonia

Recently observed patients with pneumococcal pneumonia have had the classic signs and symptoms – rigor, pleuritic pain, purulent sputum, leukocytosis, signs of consolidation on physical examination and dense segmental or lobar infiltrate on chest film. Optimal treatment is procaine penicillin G, 600,000 units injected intramuscularly every twelve hours for seven to twelve days.

When bacterial pneumonia complicates virus influenza and when gram positive cocci are seen in the sputum, either pneumococci or staphylococci is the likely cause. Such patients should be treated for staphylococcal infection until culture data is available.

Other problems in the differential diagnosis and antibiotic treatment of pneumonia have been discussed in the section on mycoplasma pneumonia.

Aspiration Lung Abscess

Aspiration lung abscess is still a problem. Postoperative aspiration or aspiration by an intoxicated or post seizure patient can mark the beginning of the necrotizing pneumonia that precedes the development of abscess. The usual stumbling block to prompt diagnosis and treatment is that the causative anaerobic organisms are not recovered in culture. They are not recovered because laboratories do not culture sputum anaerobically; the extensive anaerobic flora of the mouth would contaminate the sputum. Transtracheal or percutaneous lung puncture is the only reliable technique for anerobic culture in lung abscess. These techniques are not indicated in the usual aspiration lung abscess. Therefore, sputum cultures in aspiration pneumonia/lung abscess usually grow out normal mouth flora. If the physician remembers that the etiologic agents, the anaerobic streptococci and the respiratory tract bacteroides, are susceptible to a penicillin, he can proceed with appropriate antibiotic treatment.

Case 3. This 75-year-old man had a pyloroplasty for outlet obstruction of the stomach. Three weeks after operation he returned to the hospital with fever, cough productive of foul-smelling sputum and pleuritic chest pain in the left lower lobe. His temperature was 102°F. The leukocyte count was 16,500 with 83% polymorphonuclear leukocytes/ml. Moist rales were heard in the left lower lobe. Chest film revealed an infiltrate in the lateral basilar segment of the left lower lobe. Treatment was begun with oral ampicillin. The temperature fell to normal but follow-up chest films revealed an abscess with a fluid level in the area of the infiltrate. Treatment was changed to parenteral ampicillin 4 grams daily and postural drainage six to eight hours daily. The abscess drained and healed rapidly. After five weeks of intensive treatment, only a minimal pleural reaction was seen in the chest film.

COMMENT: Treatment of aspiration lung abscess has two important aspects – antibiotics and postural drainage. A penicillin or ampicillin should be given intravenously in generous amounts (10-20 million units of aqueous penicillin G or 4-8 grams of ampicillin every 24 hours). Should other pathogenic bacteria be present in large numbers in the initial sputum, the validity of the diagnosis should be reviewed and other explanations for the presence of other pathogenic bacteria should be considered. For example, the other bacteria present could be the consequence of recently administered antibiotics, or the "normal flora" of the patient with chronic obstructive pulmonary disease treated with antibiotics from time to time. Only rarely must another antibiotic be added to a penicillin or ampicillin in the antibiotic treatment of true aspiration lung abscess.[1]

Postural drainage is just as important as antibiotic treatment. There is no one position for postural drainage; the position depends on the location of the abscess. The patient must spend a substantial amount of time in the appropriate position. In general this amounts to about half the waking hours. The time/position relationship is dependent on the position involved and the way one uses it. For example, in postural drainage of an aspiration lung abscess in the dependent portions of the lower lobes, a patient could not lie at a 90° angle hanging over the edge of the bed for half a day, but he could hang over the edge of the bed for fifteen minutes four times a day and lie at a less acute angle most of the rest of the day. Frequent chest films must be taken to evaluate the effectiveness of the drainage and other treatment.

Endometritis

Endometritis complicates normal and complicated delivery, dilatation and curettage, other surgery involving

the uterus, and in particular – abortions. In recent years, attempts to remove intra-uterine contraceptive devices (IUD) in pregnant patients have been complicated by endometritis.

Case 4. A 28-year-old woman missed a menstrual period and consulted her physician who established that the patient was pregnant. The physician tried unsuccessfully to remove the IUD. One week later the patient developed fever, chilly feelings and dull uterine pain. Vaginal discharge and repeated rigor occurred. On admission, the patient was acutely ill with a temperature of 105°F. The uterus was painful on examination. Gram stain of the uterine discharge revealed myriads of gram negative rods. E. coli susceptible to cephalothin, chloramphenicol, colistin and many other antibiotics grew on culture of uterine discharge.

The patient was treated with intravenous cephalothin (8 grams daily) and chloramphenicol (3 grams a day). The temperature fell to normal in two days. The IUD was removed two days later. Dilatation and curettage for the removal of the dead fetus was also performed at that time. Seven days after treatment was started, colistin was substituted for cephalothin and chloramphenicol.

COMMENT: For initial treatment of endometritis, a combination of cephalothin and chloramphenicol seems most appropriate. The reasons for this choice of antibiotics are developed in some detail in the following paragraphs. Infection of the uterus is usually caused by one or more of the organisms listed in Table 4 (the aerobic gram negative flora includes E. coli, A. aerogenes, klebsiella and proteus species). Gram stains and cultures (including Novak type) should be taken but their predictive value as indicators of the etiologic agent varies considerably. For example, gram stains revealing a great number of large gram positive rods without spores strongly suggest Cl. perfringens is the infecting organism. Stains revealing gram positive cocci in pairs or occasional small chains do not differentiate between anaerobic streptococci, Group A streptococci, and staphylococci, but they do strongly suggest that the pathogen is one of these three gram positive cocci. Gram negative rods, seen on gram stain may represent aerobic or anaerobic organisms. Aerobic gram negative rods can be present as part of the normal flora or as infecting agents, whereas the anaerobic gram negative rods (bacteroides) when present are usually the cause of the infection.

When culture later reveals anaerobic streptococci, Group A streptococci, staphylococci, bacteroides, or clostridia as the predominant organism, the single most effective antibiotic for the cultured organism is substituted for cephalothin and chloramphenicol. But when E. coli, A. aerogenes, klebsiella or proteus organisms prove to be the predominant organism on culture, the single most effective antibiotic for the cultured microorganism is substituted for cephalothin, and in addition, chloramphenicol is continued for five days because I am always concerned about bacteroides not recovered in culture. If all is well after five days, I continue the single effective antibiotic to which the recovered microorganism is susceptible for five to ten more days, depending on the clinical situation and stop the chloramphenicol.

TABLE 4

COMMON PATHOGENIC BACTERIA IN ENDOMETRITIS

Microorganism	*Initial Treatment* *Cephalothin*	*Chloramphenicol*
Aerobic gram negative flora	V	V
Anaerobic streptococci	+	+
Clostridium species	+	+
Bacteroides species	V	+
S. aureus	+	V
Group A streptococci	+	+

+ = generally susceptible
V = variable susceptibility

TABLE 5

WARNING SIGNS AND SYMPTOMS OF GRAM NEGATIVE BACTEREMIA

1. Source of bacteremia.
2. Precipitating factor.
3. Low grade fever.
4. Hyperventilation.
5. Confusion.

Regarding simultaneous use of cidal and static antibiotics, when they are both employed in full doses, no clinically significant interference has ever been demonstrated except in the case of meningococcal meningitis. Therefore, when there is a demonstrated need to use a static with a cidal antibiotic as in endometritis, I do not hesitate to do so.

Chloramphenicol is used because it is the most effective and well-tested drug for all of the bacteroides species.[2] The point of this is that while the respiratory strains of bacteroides are generally susceptible to penicillins and many other antibiotics, including chloramphenicol, the gastrointestinal bacteroides especially Bacteroides fragilis are not susceptible to penicillin. B. fragilis is susceptible to lincomycin and clindamycin as well as chloramphenicol but the greatest in vitro and vivo experience is with chloramphenicol. Therefore, chloramphenicol is the drug of choice when bacteroides is suspected or present in endometritis because the bacteroides present are usually gastrointestinal strains.

Cephalothin is used for its gram positive spectrum of activity including penicillin resistant staphylococci and its effectiveness in treating E. coli, proteus mirabilis and some klebsiella infections.

BACTEREMIA

Gram negative rod

Despite innumberable papers and much discussion, there is still indecision in the diagnosis and treatment of gram negative bacteremia. The problem of the patient described below will be analyzed to suggest a reasonable approach to diagnosis and treatment.

Case 5. A 63-year-old man had had diabetes mellitus for 15 years. Two months prior to admission a transurethral resection of the prostate was performed. Recovery was uneventful until

Continued on Page 35

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Effect on PBI Determination and I^{131} Uptake: Isopropamide iodide may alter PBI test results and will suppress I^{131} uptake. Substitute thyroid tests unaffected by exogenous iodides.

Precautions: Use cautiously in persons with cardiovascular disease, glaucoma, prostatic hypertrophy, hyperthyroidism.

Adverse Reactions: Drowsiness, excessive dryness of nose, throat or mouth; nervousness; or insomnia. Also, nausea, vomiting, epigastric distress, diarrhea, rash, dizziness, weakness, chest tightness, angina pain, abdominal pain, irritability, palpitation, headache, incoordination, tremor, dysuria, difficulty in urination, thrombocytopenia, leukopenia, convulsions, hypertension, hypotension, anorexia, constipation, visual disturbances, iodine toxicity (acne, parotitis).

Supplied: Bottles of 50 capsules.

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Each white tablet contains:
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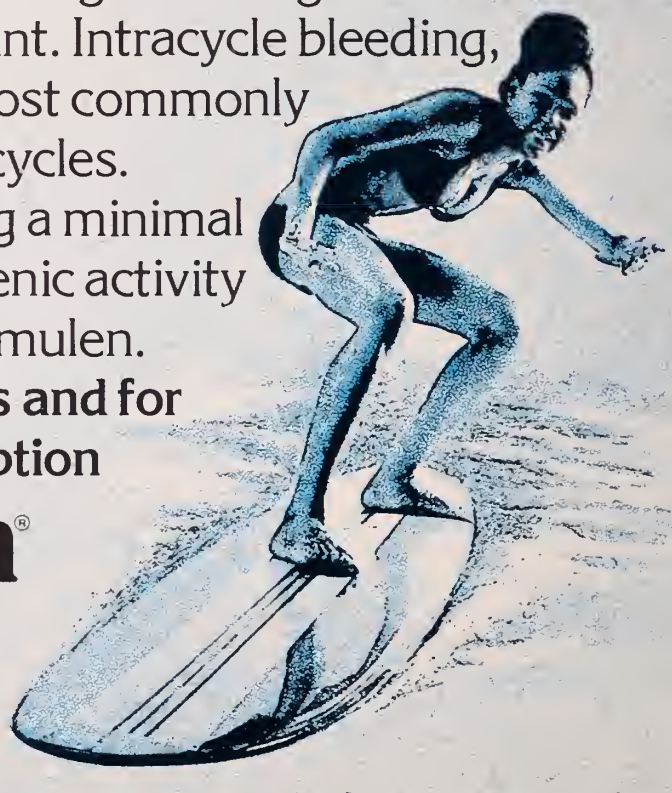
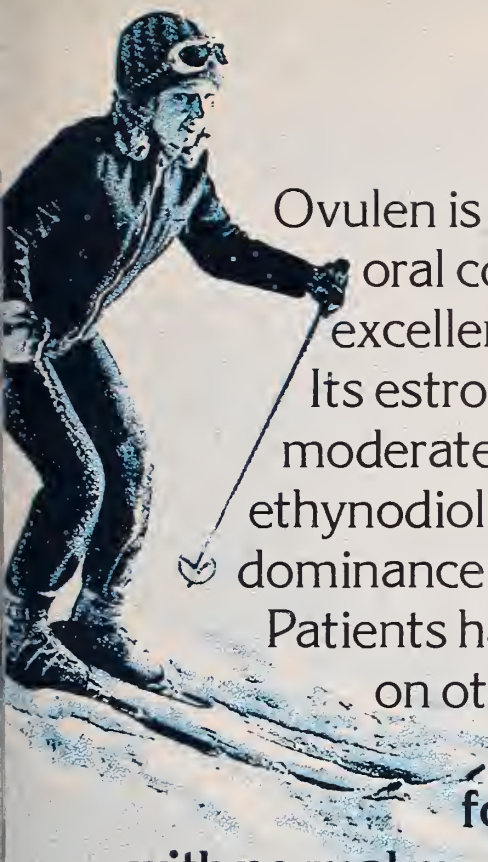


Note: Oral contraceptives are complex medicatio
They should be prescribed with care only af
reference to the prescribing information.

For brief summary of prescribing information, please see next page.



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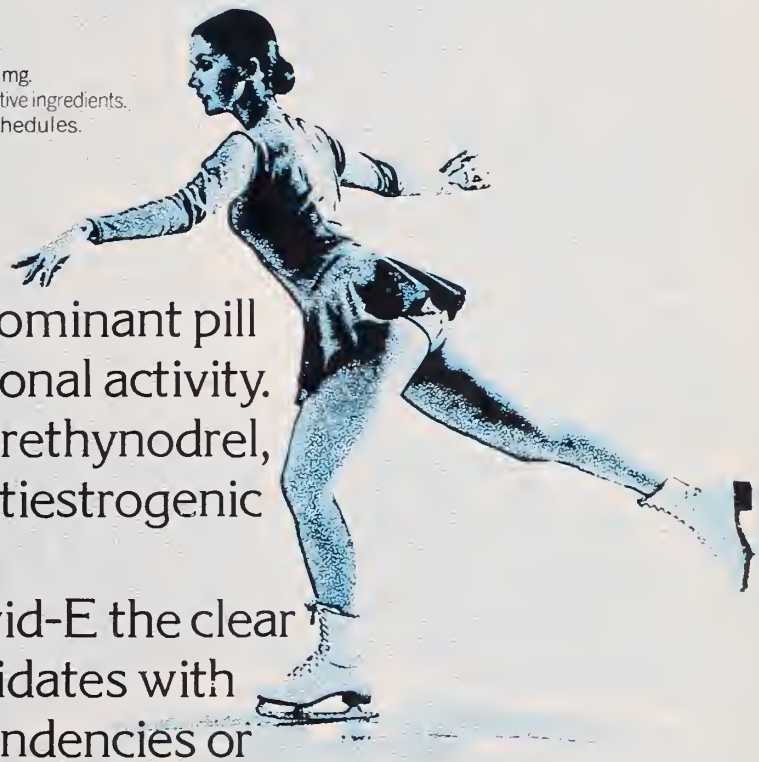
Each white tablet contains ethynodiol diacetate 1 mg./mestranol 0.1 mg.
ch pink tablet in Ovulen-28® and Demulen-28® is a placebo, containing no active ingredients.
Both Ovulen and Demulen are available in 21- and 28-pill schedules.



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Each white tablet contains
ethynodiol diacetate 1 mg / mestranol 0.1 mg.

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Each white tablet contains
ethynodiol diacetate 1 mg / ethinyl estradiol 50 mcg.

Each pink tablet in Ovulen-28® and Demulen-28® is a placebo, containing no active ingredients.

Actions – Ovulen and Demulen act to prevent ovulation by inhibiting the output of gonadotropins from the pituitary gland. Ovulen and Demulen depress the output of both the follicle-stimulating hormone (FSH) and the luteinizing hormone (LH).

Special note – Oral contraceptives have been marketed in the United States since 1960. Reported pregnancy rates vary from product to product. The effectiveness of the sequential products appears to be somewhat lower than that of the combination products. Both types provide almost completely effective contraception.

An increased risk of thromboembolic disease associated with the use of hormonal contraceptives has now been shown in studies conducted in both Great Britain and the United States. Other risks, such as those of elevated blood pressure, liver disease and reduced tolerance to carbohydrates, have not been quantitated with precision.

Long-term administration of both natural and synthetic estrogens in subprimate animal species in multiples of the human dose increases the frequency of some animal carcinomas. These data cannot be transposed directly to man. The possible carcinogenicity due to the estrogens can be neither affirmed nor refuted at this time. Close clinical surveillance of all women taking oral contraceptives must be continued.

Indication – Ovulen and Demulen are indicated for oral contraception.

Contraindications – Patients with thrombophlebitis, thromboembolic disorders, cerebral apoplexy or a past history of these conditions, markedly impaired liver function, known or suspected carcinoma of the breast, known or suspected estrogen-dependent neoplasia and undiagnosed abnormal genital bleeding.

Warnings – The physician should be alert to the earliest manifestations of thrombotic disorders (thrombophlebitis, cerebrovascular disorders, pulmonary embolism and retinal thrombosis). Should any of these occur or be suspected the drug should be discontinued immediately.

Retrospective studies of morbidity and mortality conducted in Great Britain and studies of morbidity in the United States have shown a statistically significant association between thrombophlebitis, pulmonary embolism, and cerebral thrombosis and embolism and the use of oral contraceptives. There have been three principal studies in Britain[1-3] leading to this conclusion, and one[4] in this country. The estimate of the relative risk of thromboembolism in the study by Vessey and Doll[2] was about sevenfold, while Sartwell and associates[4] in the United States found a relative risk of 4.4, meaning that the users are several times as likely to undergo thromboembolic disease without evident cause as nonusers. The American study also indicated that the risk did not persist after discontinuation of administration and that it was not enhanced by long-continued administration. The American study was not designed to evaluate a difference between products. However, the study suggested that there might be an increased risk of thromboembolic disease in users of sequential products. This risk cannot be quantitated and further studies to confirm this finding are desirable.

Discontinue medication pending examination if there is sudden partial or complete loss of vision, or if there is a sudden onset of proptosis, diplopia or migraine. If examination reveals papilledema or retinal vascular lesions medication should be withdrawn.

Since the safety of Ovulen and Demulen in pregnancy has not been demonstrated, it is recommended that for any patient who has missed two consecutive periods pregnancy should be ruled out before continuing the contraceptive regimen. If the patient has not adhered to the prescribed schedule the possibility of pregnancy should be considered at the time of the first missed period.

A small fraction of the hormonal agents in oral contraceptives has been identified in the milk of mothers receiving these drugs. The long-range effect to the nursing infant cannot be determined at this time.

Precautions – The pretreatment and periodic physical examinations should include special reference to the breasts and pelvic organs, including a Papanicolaou smear since estrogens have been known to produce tumors, some of them malignant, in five species of subprimate animals. Endocrine and possibly liver function tests may be affected by treatment with Ovulen or Demulen. Therefore, if such tests are abnormal in a patient taking Ovulen or Demulen, it is recommended that they be repeated after the drug has been withdrawn for two months. Under the influence of progestogen-estrogen preparations preexisting uterine fibromyomas may increase in size. Because these agents may cause some degree of fluid retention, conditions which might be influenced by this factor, such as epilepsy, migraine, asthma, cardiac or renal dysfunction, require careful observation. In breakthrough bleeding, and in all cases of irregular bleeding per vaginam, nonfunctional causes should be borne in mind. In undiagnosed bleeding per vaginam adequate diagnostic measures are indicated. Patients with a history of psychic depression should be carefully observed and the drug discontinued if the depression recurs to a serious degree. Any possible influence of prolonged Ovulen or Demulen therapy on pituitary, ovarian, adrenal, hepatic or uterine function awaits further study. A decrease in glucose tolerance has been observed in a significant percentage of patients on oral contraceptives. The mechanism of this decrease is obscure. For this reason, diabetic patients should be carefully observed while receiving Ovulen or Demulen therapy. The age of the patient constitutes no absolute limiting factor, although treatment with Ovulen or Demulen may mask the onset of the climacteric. The pathologist should be advised of Ovulen or Demulen therapy when relevant specimens are submitted. Susceptible women may experience an increase in blood pressure following administration of contraceptive steroids.

Adverse reactions observed in patients receiving oral contraceptives – A statistically significant association has been demonstrated between use of oral contraceptives and the following serious adverse reactions: thrombophlebitis, pulmonary embolism and cerebral thrombosis.

Although available evidence is suggestive of an association, such a relationship has been neither confirmed nor refuted for the following serious adverse reactions: neuro-ocular lesions, e.g., retinal thrombosis and optic neuritis.

The following adverse reactions are known to occur in patients receiving oral contraceptives: nausea, vomiting, gastrointestinal symptoms (such as abdominal cramps and bloating), breakthrough bleeding, spotting, change in menstrual flow, amenorrhea during and after treatment, edema, chloasma or melasma, breast changes (tenderness, enlargement and secretion), change in weight (increase or decrease), changes in cervical erosion and cervical secretions, suppression of lactation when given immediately post partum, cholestatic jaundice, migraine, rash (allergic), rise in blood pressure in susceptible individuals and mental depression.

Although the following adverse reactions have been reported in users of oral contraceptives, an association has been neither confirmed nor refuted: anovulation post treatment, premenstrual-like syndrome, changes in libido, changes in appetite, cystitis-like syndrome, headache, nervousness, dizziness, fatigue, backache, hirsutism, loss of scalp hair, erythema multiforme, erythema nodosum, hemorrhagic eruption and itching.

The following laboratory results may be altered by the use of oral contraceptives: hepatic function, increased sulfobromophthalein retention and other tests, coagulation tests, increase in prothrombin, Factors VII, VIII, IX and X, thyroid function, increase in PBI and butanol extractable protein bound iodine, and decrease in T^3 uptake values, metyrapone test and pregnanediol determination.

References: 1. Royal College of General Practitioners: Oral Contraception and Thrombo-Embolic Disease. J. Coll. Gen. Pract. *13*:267-279 (May) 1967. **2.** Inman, W. H. W., and Vessey, M. P.: Investigation of Deaths from Pulmonary, Coronary, and Cerebral Thrombosis and Embolism in Women of Child-Bearing Age. Brit. Med. J. *2*:193-199 (April 27) 1968. **3.** Vessey, M. P., and Doll, R.: Investigation of Relation Between Use of Oral Contraceptives and Thromboembolic Disease. A Further Report. Brit. Med. J. *2*:651-657 (June 14) 1969. **4.** Sartwell, P. E., Masi, A. T., Arthes, F. G., Greene, G. R., and Smith, H. E.: Thromboembolism and Oral Contraceptives. An Epidemiologic Case-Control Study. Amer. J. Epidem. *90*:365-380 (Nov.) 1969.

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Actions – Enovid-E acts to prevent ovulation by inhibiting the output of gonadotropins from the pituitary gland. Enovid-E depresses the output of both the follicle-stimulating hormone (FSH) and the luteinizing hormone (LH).

Indication – Enovid-E is indicated for oral contraception.

The *Special Note, Contraindications, Warnings, Precautions* and *Adverse Reactions* listed above for Ovulen and Demulen are applicable to Enovid-E and should be observed when prescribing Enovid-E.

Enovid-E®

brand of norethynodrel with mestranol

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Where "The Pill" Began

TABLE 6

GRAM NEGATIVE BACTEREMIA

Organisms	Initial Treatment Gentamicin and Cephalothin	OR	Kanamycin and Colistin	
E. coli	+	+	+	+
A. aerogenes	+	O	+	+
Klebsiella species	+	V	+	+
Proteus mirabilis	+	+	+	O
Indole negative species	+	O	+	O
Pseudomonas species	+	O	O	+

+ = generally susceptible
V = variable susceptibility
O = usually resistant

one week prior to admission when low grade fever, general malaise and malodorous urine were noted. Shortly after admission, the patient's fever increased. Later there was a shaking chill. Then the familiar march of events took place – tachypnea, confusion, falling blood pressure, and decreased urinary output. On physical examination, there was also tenderness in both costovertebral angles. Initial antibiotic management consisted of cephalothin, 8 grams daily, plus gentamicin, 3 mgs/kilogram/day. When Proteus mirabilis susceptible to ampicillin was identified in urine and blood cultures, parenteral ampicillin was substituted for cephalothin and gentamicin. The patient went on to make a satisfactory recovery.

COMMENT: This virulent infection probably came about as a consequence of the formation of a calcium urate bladder stone in a urinary tract infected with Proteus mirabilis. Stones commonly complicate proteus urinary tract infections and, acting as a foreign body, seem to precipitate ascending urinary tract infection. In developing gram negative shock, this patient exhibited all the warning signs and symptoms listed in Table 5.

The major organisms involved in gram negative infection originating in the urinary tract are listed in Table 6. Consideration of such things as the probable source of the bacteremia, earlier cultures taken from possible foci of the bacteremia, previous antibiotic treatment, etc., sometimes suggest the species of bacteria involved but much of the time this information is not available. Therefore, one must tentatively conclude that the patient suffers from bacteremia, probably gram negative, but with no sure indication as to the exact species of the gram negative rod involved. The principles that guide antibiotic selection and treatment of probably gram negative bacteremia are that treatment must be started immediately and that antibiotics that control the major microbial possibilities must be given from the beginning of the treatment.

Prior to the availability of gentamicin, I usually started antibiotic treatment with two bactericidal antibiotics – kanamycin and colistin (in appropriate dosage with due regard for renal function and urinary output); together they were and are effective against most of the major possibilities listed (and others that are unlisted) in Table 6. As soon as the organism was identified and the antibiotic susceptibility known, one of the drugs was stopped and the appropriate one was continued. Quite often both nephrotoxic drugs could be stopped and a cidal non-nephrotoxic antibiotic could be used (i.e., ampicillin in many E. coli and most Proteus mirabilis infections).

TABLE 7

SOURCES OF GRAM NEGATIVE BACTEREMIA

1. Urinary tract
2. Other primary gram negative infections
   A. Endometrium
   B. Peritoneum
   C. Lung
3. Blood transfusion
4. "Cut downs" and other inlying needles and catheters
5. Inadequate host defense (i.e.—<1000 polymorphonuclear leukocytes/ml circulating leukocytes.)

Since gentamicin has become available and because it is quite effective against most gram negative rods, I tend to use it alone for initial treatment in suspected gram negative bacteremia. I sometimes add cephalothin for added protection against some of the gram negatives (see Table 6) and to extend antibiotic coverage in the event that the bacteremia is not a gram negative bacteremia but is in fact a gram positive infection. Table 7 indicates that sources of gram negative infections can also be the source of gram positive infections. When culture and susceptibility data is available, the most effective and least toxic antibiotic is substituted for the combination of gentamicin and cephalothin. When blood cultures are not positive, the best guide is to treat the predominant bacterial species cultured from the probable source of the bacteremia.

Gram Negative Coccus

Neisseria meningiditis bacteremia is well known as a cause of bacteremic shock. The clinical clues are skin lesions and cerebrospinal fluid abnormalities. Gonococcal bacteremia is being observed more frequently. It can simulate N. meningiditis bacteremia but generally speaking the illness is not as severe as a meningococcus bacteremia.[3] The patient described below illustrates a probable Neisseria gonorrhoeae bacteremia.

Case 6. A 16-year-old school girl complained of fever and general malaise of two days' duration. The day of admission to the hospital, temperature rose to 104°F and 3 to 4 millimeter raised red macular lesions (some with vesicles) appeared on the body surface. Several of the lesions had black necrotic centers. When the patient arrived at the hospital, the temperature was only 102°F and she looked and felt quite comfortable. The patient had had a vaginal discharge for three months and the

Table 8

Antibiotic Dose Schedules for Severe Staphylococcal Infection

Antibiotic	*Total Daily Amount**	*Dose/Unit Time**	*Route*
Penicillin G +	24 million units	2 million/2 hours	IV
Ampicillin	8 grams	1 gram/3 hours	IV
Methicillin	12 grams	1 gram/2 hours	IV
Oxacillin	8 grams	1 gram/3 hours	IV
Cephalothin	8 grams	1 gram/3 hours	IV

*Average dose for adults – modifications are in order depending on renal function, etc.

+Penicillin G is ordinarily available as the potassium salt containing about 1.7 mEq/1 million units (also manufactured as the sodium salt).

cervix was slightly tender. Laboratory exam revealed a 17,200 white count with 90% polys. Lumbar puncture revealed normal cerebrospinal fluid. Cultures of vaginal discharge revealed Neisseria gonorrhoeae.

Comment: On admission it was thought that the likely diagnosis was Neisseria gonorrhoeae bacteremia and the patient was treated with 20 million units of penicillin G intravenously every 24 hours. Other diagnostic possibilities included Neisseria meningiditis, staphylococcal and pseudomonas bacteremia. There was no likely source for the pseudomonas so this possibility was not treated. And although the patient had no heart murmurs and no apparent source of staphylococcal infection, the first two days she was also treated with methicillin 12 grams intravenously daily. The methicillin was stopped as soon as blood cultures revealed no growth (in staphylococcal bacteremia, staphylococci almost always grow out in blood cultures in several days).

Multiple cultures in this patient revealed no growth. In the absence of the positive cultures, an absolute diagnosis of Neisseria gonococcemia cannot be made. However, there was strong clinical evidence and some laboratory backing to support the clinical diagnosis of gonococcemia.

Staphylococcal Sepsis

Problems are still encountered in the diagnosis and treatment of major staphylococcal sepsis. A major problem in diagnosis of staphylococcal sepsis is illustrated in the patient cited below. The point to remember is that major staphylococcal sepsis can begin as a low grade febrile disease without localizing signs and symptoms. This is especially common in such staphylococcal infections as pyemic staphylococcal pneumonia, joint infections particularly in the hip and infection of the lumbar vertebrae.

The problem in therapy is not lack of effective antibiotics, for penicillin G is as effective as ever against staphylococci that do not elaborate penicillinase and penicillinase resistant penicillins (methicillin et al) are effective against penicillinase producing as well as non penicillinase producing staphylococci. For these reasons and because approximately 40% of non hospital acquired and virtually all hospital acquired staphylococci infections are penicillin G resistant, all serious staphylococcal infections should be treated initially with a penicillinase resistant penicillin. The therapeutic problem is simply that this is not being done. Some physicians start treatment with penicillin G as well as a penicillinase resistant penicillin. This is satisfactory as long as full staphylococcal doses of both are given, but there is no convincing evidence that staphylococci susceptible to penicillin G cannot be treated equally well by adequate doses of a penicillin resistant penicillin. However, penicillin G is less expensive and it should be substituted for methicillin if susceptibility studies indicate that the staphylococcus is susceptible to penicillin G.

Case 7. This 12-year-old school girl complained of low grade fever, malaise, and nausea and vomiting of two days' duration. Physical examination and complete blood count revealed no abnormalities. At follow-up two days later, there was minimal pain on movement of the right hip, fever of 103°F, and the leukocyte count was 16,500 ml. On aspiration of the hip joint, bloody, slightly purulent material was obtained. Gram stain revealed gram positive cocci singly and in pairs. Cultures grew out penicillin resistant S. aureus. The patient was treated with methicillin intravenously for six weeks. After the first week of treatment, the dosage was reduced from 16 to 12 grams daily. Six weeks rather than four weeks therapy was used because the patient had a low grade fever during the first two weeks of treatment. Repeated aspiration of the hip joint revealed no bacteria and no other non-infectious cause of fever was found.

Comment: Doses of antistaphylococcal antibiotics required for serious staphylococcal infections are listed in Table 8. In addition to large doses administered for long periods of time, antistaphylococcal drugs should be given at frequent intervals. There is reason to believe that an effective drug level must be present for about 20 out of every 24 hours in treating serious staphylococcal infections.[4]

Peritonitis

Peritonitis poses problems in devising antibiotic therapy because in relatively localized peritonitis often no bacteria are recovered, whereas in diffuse peritonitis even with the results of cultures at hand, one worries whether or not the bacteria obtained are representative of all areas of the infection. No matter which of the two situations obtain, it is necessary to treat the infection for three or four days before culture results and susceptibility tests are available. In selecting the antibiotics for treating peritonitis, I use much the same approach as in treating endometritis (Table 9 lists the common pathogens in peritonitis) and start treatment with cephalothin and chloramphenicol. Note that enterococci are not listed. There is no doubt the enterococci are frequently present in peritonitis because they are part of the normal bowel flora, but there is considerable doubt about their pathogenicity in the peritoneum. Similarly, of the aerobic gram negative bowel flora only E. coli and A. aerogenes are commonly involved. Pseudomonas is rarely a cause of peritonitis.

Table 9

Common Pathogenic Bacteria in Peritonitis

Microorganism	*Initial Treatment Cephalothin*	*and Chloramphenicol*
Aerobic gram negative flora	V	V
Anaerobic streptococci	+	+
Clostridium species	+	+
Bacteroides species	V	+
S. aureus	+	V

+ = generally susceptible
V = variable susceptibility

Case 8. A 13-year-old camper was referred with a diagnosis of peritonitis. Six weeks prior to admission appendectomy was performed. The patient recovered and came to a Maine summer camp. For three or four days prior to admission, the patient had intermittent abdominal pain and gradually developed signs and symptoms of acute peritonitis. After preparation, surgical exploration revealed diffuse peritonitis; the source was found to be a perforation in the distal ileum. The appendectomy site appeared to be well healed. Gram stain of the peritoneal fluid revealed gram negative rods. Antibiotic treatment was begun with cephalothin (8 grams/day) and chloramphenicol (3 grams/day) given intravenously. Cultures revealed Aerobacter cloacae* susceptible to chloramphenicol, cephalothin and to other antibiotics including colistin. When the cultures were returned, both antibiotics were continued; chloramphenicol because of the concern over undetected bacteriodes. The patient made a good recovery, complicated only by a relatively superficial wound infection. After 7 days, chloramphenicol was discontinued because the signs of peritonitis had disappeared promptly. Colistin was substituted for cephalothin after 10 days and continued for 7 more days in treating the complicating wound abscess.

Comment: There is no panacea for diffuse peritonitis but using the approach described I have observed excellent results in a series of 20 patients. Though there has been considerable morbidity, the only patients who died were moribund prior to treatment or had additional life-threatening illnesses. Treatment with the two antibiotic, cidal and static combination of cephalothin and chloramphenicol has proved most effective in controlling the varied causes of diffuse peritonitis. Chloramphenicol is always used with a good deal of reluctance but when one is trying to give the most effective antibiotic for bacteriodes, there is no other choice at the present time.‡ The reticulocyte count should be followed closely and an attempt made to limit the use of chloramphenicol to seven to ten days.[5]

*Formerly A. aerogenes. However in the discussion and tables in this text the newer taxonomic system of Ewing has not been used.

‡Clindamycin is a promising substitute.

Summary

The case presentations emphasize the need for accurate clinical delineation of the illness. Thorough appraisal carries with it the need to evaluate the etiology – in this context this means the microorganisms most likely to cause the illness. Finally, having considered the usual antibiotic susceptibility of various microorganisms that could be involved, one can make a rational selection of antibiotics.

The principles of antimicrobial therapy developed here are generally accepted; however, exception can and probably will be taken to various individual statements. For those interested in a more extensive discussion of antibiotic therapy, a good general reference is the Handbook of Microbial Therapy published by The Medical Letter.[6]

References

1. Abernathy, R. S.: Antibiotic Therapy of Lung Abscess; Effectiveness of Penicillin. Dis. Chest, 53: 592, 1968.
2. Bardner, S. J., Koenig, M. G., Treanor, L. L. and Gordman, J. S.: Antibiotic Susceptibility Testing of Bacteriodes. Antimicrob. Agents Chemother., 2: 57, 1972.
3. Abu-Nassar, H., Hill, N., Fred, H. L. and Yow, E. M.: Cutaneous Manifestations of Gonococcemia. Arch. Intern. Med. 112: 731, 1963.
4. McDermott, W.: Chemotherapy of Microbial Diseases in Bacterial and Mycotic Infection of Man. Duhos, R. J. (ed). Philadelphia, Lippincott, 1958, p. 711.
5. Scott, J. L., Finegold, S. M., Belkin, G. A., and Lawrence, J. S.: A controlled double blind study of the hematologic toxicity of chloramphenicol. New Eng. J. Med. 272: 1137, 1965.
6. Handbook of Antimicrobial Therapy. The Medical Letter. 14: 3-63, 1972. New York, 1972.

COMBINED SCALP AND SKULL RESECTION FOR BASAL CELL CANCER

Continued from Page 31

2. Conley, J.: Cancer of the middle ear. Ann. Oto. Rhin. Laryng. 74, 555-572, June 1965.
3. Hanna, D. C., Richardson, G. S., and Garsfind, J. C.: A suggested technique for resection of the temporal bone. Am. J. Surg. 114, 553-558, Oct. 1962.
4. Terz, J. J., Alksne, J. F., and Lawrence, W., Jr.: En bloc resection of the pterygoid region in the management of advanced oropharyngeal carcinoma. Surg. Gyn. & Ob. 130, 349-352, Feb., 1970.
5. Edgerton, M. T., and Snyder, G. B.: Combined intracranial-extracranial approach and use of the two-stage split flap technique for reconstruction with craniofacial malignancies. Am. J. Surg. 110, 595-602, Oct. 1965.

A Team Approach to Renal Dialysis in a Community Hospital

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Mary Hardwicke, R.N. and Penny Bresnick, R.N.

Purpose

To report our experience with the team approach to hemodialysis consisting of nursing team made up of trained registered nurses, to carry out the actual dialysis under the supervision of physicians specializing in internal medicine.

Methods

Between the years of 1967 and 1969 in the Waterville area, 55 patients expired of uremia. Based on study of our medical records, several of those patients appeared to have been candidates for hemodialysis, and two of them were actually referred to other centers where dialysis could be carried out. For this reason, it was felt that some effort to establish dialysis in our own area might well raise the level of care for certain patients with uremia. However, there was no experience with this modality of treatment in the area. It was therefore determined to approach this problem from the point of view of a nursing organization using techniques similar to coronary care. One physician visited a dialysis unit, an artificial kidney was obtained, and during the month of March 1970, a physician and 6 nurses began concomitant training consisting first of the assembly of the dialyzer which was a Travenol® twin coil. Development of the protocols for dialysis, dry runs were established and animal dialysis was accomplished by the end of the month. In the month of April, the first patient was started on hemodialysis. Since that time, 19 patients have been treated. Of these, one patient has received successful transplantation in Boston. Two patients with acute tubular necrosis have been restored to normal function. One patient with acute tubular necrosis expired after a period of successful dialysis. This patient was lost to follow up and a recurrence of renal failure developed. The patient was later admitted in terminal condition with uremic pericarditis. Experiences of all patients are summarized in Table 1.

Summary

As of June 20, 1972, a total of 19 patients have received a total of 320 individual dialysis treatments, and over 1,800 hours of dialysis experience has been acquired. There have been no significant complications or deaths attributable to dialysis. It is planned to move the dialyzer from a single small room which it now occupies to a larger room so that dialysis on two patients may be carried out at the same time. At the present time, the cost of hemodialysis is $150 per treatment. Physicians are remunerated on the basis of their usual daily minimal fee unless they are required for a prolonged time with the patient, for which they receive their usual customary fees for a prolonged detention on an hourly basis. It has been unusual for physicians to have to spend much time with patients during chronic dialysis, and the whole procedure is often carried out by the nurse. The physician is not required to be present but should be available in the event of difficulties.

At the present time, there are 6 nurses who are qualified to carry out hemodialysis, and two have extensive experience. In the event of an emergency, dialysis can be carried out on short notice. In addition, there are three internists who are qualified to supervise dialysis. All patients in the chronic dialysis programs have received arterial venous fistulas. Patients on short-term dialysis have been dialyzed by cannulation or with Scribner shunts which have subsequently been removed. All vascular surgery is done by general surgeons with vascular experience.

Conclusion

A team approach to dialysis with close physician-nurse cooperation provides a feasible solution to the practical and economic problems in community hospitals serving rural areas. Hemodialysis can be carried out within a reasonable cost by nurse technicians close to the patient's place of residence with minimal supervision. This type of program serves not only to make dialysis available to people who could not be dialyzed because of long distances of communication and should in the future serve as a relief mechanism for larger transplantation centers so that patients who need maintenance dialysis can be dialyzed away from the center.

Finally, these procedures can be carried out at reasonable cost provided that proper motivation of physicians and nursing staff and hospital can be developed.

TABLE 1

Age	Sex	Number of Treatments	Home	Complications	Diagnosis	Comments
59	M	11	Madison, Me.	0	Acute tubular necrosis.	Developed acute tubular necrosis following Loridine® 4 grams per day for peritonitis complicating exploratory laparotomy. Discharged to be followed by referring physician. Lost to follow-up until readmitted 4 months later comatose with uremic pericarditis. Expired of ventricular fibrillation and congestive failure. Post-mortem: yes.
69	M	2	Corinna, Me.	0	Obstructed duodenal ulcer. Postoperative anemia.	Developed azotemia and mental confusion following surgery for obstructed duodenal ulcer. Previous history of chronic obstructive lung disease. Finally expired of cardiopulmonary failure. Post-mortem: acute tubular necrosis, carcinoma of kidney, chronic obstructive pulmonary emphysema and coronary artery disease.
39	M	12	Fairfield, Me.	0	Diabetes, diabetic nephropathy, secondary hypertension, congestive heart failure.	Juvenile diabetes with Kimmelstiel-Wilson syndrome. Symptoms relieved by dialysis but finally expired due to acute coronary thrombosis and cardiac failure. Post-mortem also showed uremic pericarditis and classical diabetic nephropathy.
64	M	3	N.Y. City; Great Pond, Me.	0	Chronic renal failure, hyperkalemia.	Admitted and found to be uremic and hyperkalemic while on vacation from N.Y. City. No previous history of renal disease. Died of hyperkalemia while another hyperkalemic patient being dialyzed. No Post-mortem. Permission refused.
38	F	10	Sullivan, Me.	0	Acute renal failure.	Developed acute brain syndrome following delivery. Thought to have brain tumor. Refused to neurosurgery and found to have azotemia, proteinuria, and r.b.c. casts. Post-mortem: multiple cerebral and renal emboli with endocarditis.
68	M	1	East Wilton, Me.	0	Chronic renal failure.	Admitted with intestinal obstruction with elevated BUN. Explored and developed postoperative oligemia. Died of cardiopulmonary failure. Post-mortem: chronic pyelonephritis and chronic obstructive pulmonary emphysema.
19	F	4	Madison, Me.	0	Acute tubular necrosis due to shock from hemorrhage due to placenta previa.	Has had complete recovery.
45	M	18	Waterville, Me.	0	Acute cortical necrosis	Has had successful transplantation.
66	M	16	Madison, Me.	0	Diabetic glomerulosclerosis.	Symptoms of nausea, dyspnea. Found to have recent coronary thrombosis, congestive heart failure, classical glomerulosclerosis on post-mortem.
17	M	6	Waterville, Me.	0	Shot gun wound of abdomen. Renal failure.	Self-inflicted shot gun blast to abdomen requiring resection of large portion of bowel, 1 ureter destroyed. Irreversible shock. Acute tubular necrosis, on post-mortem and peritonitis.
66	M	3	Waterville, Me.	0	Renal tubular necrosis.	Intestinal obstruction due to incarcerated hernia. Followed by multiple fistulae and shock septic type with peritonitis and renal failure. Expired: Post-mortem.
58	M	4	Skowhegan, Me.	0	Acute tubular necrosis due to Loridine shock.	Avulsion with fracture right arm in traction shaft. Developed acute tubular necrosis. Arm amputated. Discharged. Renal function normal.
65	M	40	Livermore Falls, Me.	0	Chronic glomerulonephritis.	Started on dialysis in January, 1971. Comes in weekly. On chronic dialysis. Doing well.
60	M	43	Abbott, Me.	0	Chronic glomerulonephritis.	Chronic glomerulonephritis with total cortical atrophy hypertensive heart disease. Multiple pulmonary emboli. Post-mortem: Yes.
62	F	161	Stratton, Me.	0	Chronic glomerulonephritis.	Admitted weekly for dialysis. Doing fair.
76	F	11	Waterville, Me.		Nephrosclerosis.	Expired of ruptured aortic aneurysm. No post-mortem.
69	F	2	Oakland, Me.		Acute tubular necrosis.	Developed peritonitis from acute cholecystitis following surgery. Treated with Loridine and Garamycin®. Off dialysis. Still in polyuric phase.
66	M	6	Pittsfield, Me. 25 miles	0	Acute proliferative glomerulonephritis.	History chronic obstructive lung disease. Developed sudden elevated BUN. Post-mortem chronic obstructive lung disease, acute proliferative glomerulonephritis.
58	M	2	Buffalo, N.Y.		Glomerulonephritis.	Treated while attending convention in Augusta.

Angiographic Features of Ruptured Abdominal Aortic Aneurysm

John T. Chen, M.D.

Because of the increase of the aging population, abdominal aortic aneurysm has become a relatively common disease in recent years. Most of the expanding aneurysms end up in spontaneous rupture anteriorly. This catastrophe is often of such rapidity that many patients succumb before they have time to reach the hospital.

The clinical diagnosis of large or ruptured abdominal aneurysm is usually not difficult especially if a calcified wall can be demonstrated on plain films of the abdomen. A smaller percentage of an aneurysm will rupture posteriorly into the retroperitoneal space and form a hematoma. This situation is generally somewhat less acute but often elicits confusing symptoms and signs.

The following case presentations illustrate a few of the varieties where preoperative abdominal aortography was useful in confirming or establishing the diagnosis.

Case Presentations

Case 1:

P.M.: This 63-year-old male was referred to Thayer Hospital for a suspected leaking abdominal aortic aneurysm of one day's duration. A small abdominal aneurysm was detected 18 months previously but because of several previous attacks of coronary artery disease and strokes, an elective resection was deferred.

At time of admission, patient had intermittent attacks of severe right flank pain, anuria and cardiac arrythmia. Due to patient's coronary artery disease and poor condition, it was decided to perform an abdominal aortogram to confirm the diagnosis and also to evaluate the status of renal arteries. The aortogram which was performed with a small volume of contrast media (Fig. 1) demonstrated a projection (arrow) of the lumen on the right consistent with the point of rupture and an area of filling defect by thrombus. The renal arteries were normal.

The patient was operated on after the PVC was controlled by I.V. lidocaine drip. A replacement Dacron prothesis was utilized following excision of the ruptured aneurysm. The patient had a poor postoperative course, developed hypotension, repeat myocardial infarction, cerebral and femoral embolism, and expired thirty hours following surgery.

Case 2:

R.C.: This 67-year-old male was studied as an outpatient because of an enlarging left abdominal mass of approximately four months' duration. While there was some pulsation to the mass, it was relatively feeble and there was no evidence of calcifications. The left psoas margin was not obliterated. There was displacement of the left colon and the left ureter throughout its course (Fig. 2), and an abdominal aortogram was done to differentiate between aneurysm and retroperitoneal mass lesion. A large sacular aneurysm (Figs. 3A and 3B) was found at aortography extending to the left dome of the diaphragm. The aorta proximal to the aneurysm was not visualized due to inability to pass the catheter beyond the aneurysm. It was thought this was a case of impending rupture and it was resected despite patient's history of three previous attacks of myocardial infarction. The patient recovered from the surgery but suffered a fatal attack of myocardial infarction eight months later.

Fig. 1. The abdominal aorta below the renal arteries is outlined by dotted lines. The arrow points to the place of rupture. The aorta is displaced to the left due to large thrombus along the right wall of the aneurysm.

Fig. 2. The left kidney and ureter are laterally displaced with a soft tissue density occupying the entire left upper two-thirds of the abdomen.

Fig. 3A. AP view outlines the aneurysm occupying the left mid and upper abdomen.

Fig. 3B. Oblique projection demonstrates aneurysm to be anterior and lateral to the upper abdominal aorta.

Fig. 4. The right ureter is laterally displaced with obliteration of the right psoas margin.

Fig. 5. Aortogram shows the large aneurysm and a somewhat triangular pouch on the right in the area of hematoma.

Fig. 6. The aneurysm (A) and vena cava (IVC) are clearly demonstrated in conjunction with a large fistula (arrows).

Case 3:

T.M.: A 70-year-old male was referred to the Neurosurgical Service because of chief complaint of femoral neuritis and questionable disc herniation. The onset dated back about two months prior to admission with increase in severity and back pain. An area of ecchymosis was found at the right groin with edema of the right inguinal region. There was also considerable tenderness in the right lower quadrant. The right ureter was displaced laterally on IVP (Fig. 4) with obliteration of the psoas margin. A leaking abdominal aortic aneurysm was suspected in view of the hemorrhage into the right groin. This was verified by subsequent aortogram (Fig. 5). The patient recovered from surgery and was discharged two weeks later.

Case 4:

H.H.: This 62-year-old male was admitted to the hospital with the chief complaint of right CVA pain of about one week's duration. No definite abdominal mass was felt on admission but there was a loud systolic bruit and moderate tenderness over the right side of the abdomen. The IVP was normal which ruled out the possibility of ureteral calculus. A subsequent aortogram (Fig. 6) showed an abdominal aneurysm which had ruptured into the inferior vena cava (arrows). In the meantime, there was a drop in patient's diastolic blood pressure and a pulsating mass could be felt at mid abdomen. It became apparent that this patient developed a small A-V fistula in the past ten days which was enlarging rapidly. The aneurysm was resected with a closure of the rupture at the vena cava followed by replacement prothesis. He had an uneventful postoperative course and is now living and well two years following surgery.

Normally, abdominal aortic aneurysm does not require aortography for diagnosis; however, in cases with atypical findings this could be useful for a definitive preoperative diagnosis. It is noteworthy that three of these cases had ruptured to the right side, not as rare as generally believed. The risk of rupture or dislodging of thrombus by aortography is minimized with the use of safety J guide wire and pigtail catheter.

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Clinical Applications of the Carotid Sinus Reflex

Stephen F. Vatner, M.D.*

For the past half century, the carotid sinus reflex has been the focus of intense physiological interest and investigation. This fundamental cardiovascular reflex influences and regulates arterial pressure, heart rate, myocardial contractility, and peripheral vascular resistance. Recently, exogenous stimulation of this reflex has been employed as a diagnostic and therapeutic tool.

The carotid sinus is a dilatation of the internal carotid artery near its origin at the bifurcation of the common carotid artery. Afferent information from the carotid sinus is relayed to the cardiovascular centers in the brainstem via the carotid sinus nerve, a branch of the glossopharyngeal. The efferent loop of the carotid sinus reflex comprises both sympathetic and parasympathetic pathways to the heart which influence heart rate and myocardial contractility and sympathetic pathways to the adrenal medulla and the peripheral arterial and venous vasculature. The reflex functions as a negative feedback system, i.e., an increase in arterial pressure increases afferent nerve traffic to the cardiovascular center, which in turn reflexly reduces peripheral vascular resistance, heart rate, myocardial contractility, and circulating catecholamines; all of which tend to return arterial pressure to control. Carotid sinus hypotension produces opposite results. A similar reflex arises from a pressure sensing mechanism located in the aortic arch. These reflexes comprise the major baroreceptor systems. The baroreceptors serve to maintain arterial pressure and cardiovascular dynamics relatively constant.

Excessive activation of the carotid sinus reflex may produce carotid sinus syncope. Susceptible patients with a hyperactive carotid sinus reflex are therefore prone to episodic hypotension and syncope; characteristically in elderly men especially, those who wear high, tight collars, and young women just prior to menstruation. Patients afflicted with Takayasu's syndrome, a non-specific arteritis affecting the subclavian and carotid arteries of young women, are susceptible to carotid sinus syncope.

One established therapeutic and diagnostic maneuver involving the carotid sinus is differentiation of supraventricular from ventricular arrhythmias and the interruption of paroxysmal atrial tachycardia by manual carotid sinus massage. Massage simulates arterial hypertension and results in a reduction of sympathetic tone and a simultaneous increase in vagal tone to the heart. Thus, automaticity decreases, conduction slows and the tachycardia is frequently abruptly terminated.

Carotid sinus massage has several potential pitfalls. The technique cannot be applied to all patients since carotid blood flow may be compromised in the presence of coexisting cerebral vascular disease. Syncope, cardiac standstill due to excessive vagal inhibition of the sino-atrial or AV nodes; dislocation of an arterial thrombus or plaque with consequent cerebral embolism are potential risks that can be minimized by careful monitoring of heart rate and limitation of duration of compression.

The carotid sinus nerves can be activated by a new technique now utilized for interrupting disabling, recurrent attacks of paroxysmal atrial tachycardia. The baroreflex is activated by electrical stimulation of the carotid sinus nerves, again, like manual stimulation, stimulating the afferent limb of the reflex arc and tending to slow the heart rate and interrupt the attack by decreasing sympathetic tone and increasing in vagal restraint. At operation, electrodes are implanted bilaterally on the carotid sinus nerves and a radio-frequency receiver attached to the electrodes is placed in a subcutaneous pocket. After the recovery from operation, the patient activates a radiofrequency pacemaker‡ to stimulate the carotid sinus nerves electrically via the receiver-electrode circuit. Thus, the reflex can be activated immediately by the patient as needed, stimulation can be discontinued abruptly, and stimulation may be initiated safely and repeatedly.

Carotid sinus massage has been used as a tool in the diagnosis of angina pectoris for many years. Recently, carotid sinus nerve stimulation has been employed also in the therapy of angina pectoris by Braunwald and co-workers. Angina pectoris results from a disparity between the oxygen requirements of the myocardium and oxygen availability via myocardial blood supply. Electrical stimulation of the carotid sinus nerves tends to diminish the myocardial oxygen requirements primarily by reducing arterial pressure, left ventricular afterload and myocardial wall tension, while it reduces heart rate and myocardial contractility to a lesser extent. In addition, carotid sinus nerve stimulation produces reflex coronary

Continued on Page VII

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Prepared by the Maine Heart Association for this Journal.

‡Medtronic, Inc., Minneapolis, Minnesota.

NEW REVIEW PROVISIONS IN H.R. 1

H.R. 1, the health regulations bill, was recently signed by President Nixon. An important amendment to this bill is the provision for Professional Standards Review Organizations (PSRO).

Two major factors have influenced legislation to establish PSRO's:

1. Increased unit cost of services
2. Increased number of services

Since increased cost can be attributed to inappropriate utilization, significant emphasis will be placed on restructuring the utilization review process. Recent estimates of the cost of the Medicare Hospital Insurance Program indicate an over-run of 1967 estimates of $240 billion over a 25-year period.

Establishment of PSRO's

The amendment reflects tentative designation of PSRO areas throughout the country by 1974. Each area is either geographic or medical service area terms would generally include a minimum of 300 participating physicians. The Secretary of HEW would provide prototype plans of organization and operation to prospective PSRO's in each area. The prototypes are to be developed in consultation with proposed PSRO's and various organizations presently operating comprehensive review mechanisms.

Proposals submitted to the Secretary of prospective PSRO's would be made available, on request, to appropriate concerned organizations and individuals who, in turn, would be free to submit to the Secretary such comments on the proposal as might assist in evaluation of the prospective PSRO. A National PSRO would monitor on a regular and continuing basis the performance of the organizations selected.

Priority in designation as a PSRO would be given to organizations established at local levels representing substantial numbers of practicing physicians who are willing and believed capable of progressively assuming responsibilities for overall continuing review of institutional and outpatient care and services.

Participation in a PSRO would be voluntary and open to every physician in the area.

Physician organizations or groupings would be completely free to undertake or to decline assumption of responsibilities of organizing a PSRO. If they decline, the Secretary would be empowered to seek alternative applicants from other medical organizations, state and local health departments, and medical schools; and failing all else, carriers and intermediaries or other health insurers.

Responsibilities of PSRO's

The PSRO would be required to regularly review provider and practitioner profiles of care and service and other data to evaluate the necessity, quality, and appropriateness of services for which payment may be made under the Medicare and Medicaid Programs.

The PSRO would also analyze the pattern of services rendered or ordered by individual practitioners and providers and to concentrate on situations where unnecessary, substandard or inappropriate services exist or occur.

The PSRO would have authority to approve the medical necessity of all elective hospital admissions in advance, solely for the purpose of Medicare-Medicaid payment.

It is the Committee's opinion that government should not have to review medical determinations unless the medical profession evidences an unwillingness to properly assume the task.

Throughout the amendment, references are made to PSRO's working with and utilizing organizations presently operating effective review mechanisms. Although final determinations may rest with the PSRO's, delegation of some functions will continue to go to utilization review committees and intermediaries.

The amendment states that a PSRO would be required to acknowledge and accept in whole or part, an individual hospital's own review of admissions and need for continued care, on a hospital-by-hospital basis where it is determined that a hospital's "in-house" review is effective. In a situation where a hospital's in-house review is not adequate, the PSRO would be responsible for reviewing attending physician's certifications of need for continued hospital stay.

While the final structure of the PSRO may be modified as the organizations are developed, we can be assured that effective utilization review, a strong peer review system, and intermediary activity are essential and will all play a role in the PSRO.

Necrology

Gerald H. Donahue, M.D.

1908-1972

Dr. Gerald H. Donahue, 64, of Presque Isle, Maine, died unexpectedly at a Presque Isle hospital on November 25.

He was born in Johnston, New Hampshire on October 16, 1908, son of Michael Stewart and Rose Ann Donahue. Dr. Donahue was graduated from Bowdoin College in 1931 and received his medical degree from McGill University Faculty of Medicine in 1936. He took postgraduate courses at the Newton Hospital and the Children's Memorial Hospital and had practiced in Presque Isle since 1938, except for service in the U.S. Army during World War II from 1941 to 1945, serving with the Maine Medical Center Unit in the European Theater for three years.

Dr. Donahue was a member of the Aroostook County Medical Society, the Maine Medical Association and the American Medical Association. He was also a member of the Presque Isle School Board from 1951 to 1963, serving as chairman of the Board for 10 years; organized the Presque Isle Alcoholic Anonymous group and was founder of the Kiwanis Child Health Clinic; was a member of the Presque Isle Rotary Club and a member of the Board of Directors of the Maine Cancer Society. He was the first recipient of the Community Service Award given annually by the Live Wire Club of the Presque Isle Chamber of Commerce.

Surviving are his widow, Geraldine Donahue of Presque Isle; a son, Michael of Levant; three daughters, Mrs. John French of Hampden, Mrs. John Paterson of Hallowell and Miss Rose Ann Donahue of Presque Isle; a brother, Clement L. Donahue, M.D. of Caribou; two sisters, Mrs. Philip Crichton of Mars Hill and Mrs. Roscoe Libby of Hinsdale, Illinois; and eight grandchildren.

News, Notes and Announcements

Pediatric ENT Course

The Otolaryngology Department of Children's Hospital Medical Center, Boston, Massachusetts, will present a course dealing with clinical areas of pediatric ENT. This course, designed expressly for pediatricians, will be held on Saturday, May 5, 1973 at the Jimmy Fund Auditorium of the Children's Cancer Research Foundation. In addition to the prepared lectures, members of the audience will be able to present diagnostic or management problems from their own practice. Formal lectures will be as follows:

Charles Ferguson, M.D.: Bronchoscopy and Bronchograms
Frank Ritter, M.D.: Sinusitis in Children
George Gates, M.D.: Facial Paralysis in Children
Maurice Miller, Ph.D.: Practical Hearing Tests for the Office
Joel Bernstein, M.D.: Serous Otitis Media
Greer Edwards, M.D.: The Draining Ear
Jan Beekhius, M.D.: Cosmetic Problems of the Nose
Marshall Strome, M.D.: Croup and Acute Epiglottitis
Burton Jaffe, M.D.: Management of the Lump in the Neck

Burton Jaffe, M.D. will moderate. Tuition is $30.00. Further information may be obtained by writing Iris Shur, coordinator, c/o Department of Otolaryngology, Children's Hospital, 300 Longwood Avenue, Boston, Mass. 02115.

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Through Examination

Aleta, Elenita F.; Aramendia, George; Au, Shiu-Cheong; Ayuyao, Adolfo M.; Balinberg, Edmond B.; Bana, Dhirendra S.; Banaag, Carolina G.; Becil, Hector M.; Bhuta, Ishwarlal; Brar, Manjit I. S.; Brezler, Mario R.; Carter, Anthony P.; Chang, Nan-Hsiung; Chen, Jai-Shong; Chillar, Ram K.; Chiong, Ignacio A.; Choi, Kyoung H.; Choi, Ock S.; Chokshi, Deenbandhu S.; Cruz, Ernesto B.; Doshi, Navnitrai C.; Entin, Erik J.; Feldbau, Gary A.; Fu, Cheng S.; Garces, Sergio M.; Gajic, Slobodan; Garg, Shankar L.; Genovesi, Michael G.; Naghshineh-Ghofrany, Fereshteh; Han, Chung W.; Hanna, Wagih F. A.; Haq, Jamshedul; Henriques, Edgar S.; Hindi, Adib; Husain, Musa K.; Hyon, Won; Hyun, Jong D.; Jamshidnejad, Javad; Jothi, Rishyur K.; Jozefowicz, Thaddeus H.; Kang, Shin Y.; Kantharaj, A. Joseph; Kellan, Carl; Khair, Gamil Z.; Kim, Chang C.; Kim, Joon W.; Kumar, Nirmal A.; Kumar, Sourirajulu A.; Kumari, S. Sheila; Laczkowski, Andrzej W.; Lee, Chang B.; Lee, Tae A.; Lee, Tay B.; Lerner, Nolan E.; Mailepors, Harold; Mukhopadhyay, Madan G.; Naffis-Sahely, Daryoush; Nevrekar, Lila N.; Newman, Richard A.; Pak, Undong; Park, Byung W.; Patel, Pratap P.; Pavlica, Dusan; Pierce, Harrison; Prendergast, John C.; Purpora, David; Raja, Naseer M.; Reddy, Kolli P.; Rhee, Yong W.; Salerno, Hugo R.; Sanchez, Eduardo A.; Sarmiento, Reynaldo D.; Sekaran, S. Kamala; Shah, Harnishkumar V.; Shah,

Jagdish R.; Singh, Bijai B.; Singh, Gurbachan; Singh, Shivdev; Song, Kwan H.; Srinavasan, Sethurama; Sun, Tsieh; Taleghany, Ahmad S.; Tandon, Vipinkumar M.; Tarar, Riaz A.; Teodorescu, Marin; Toback, Neil E.; Trujillo, Manuel; Veltri, Frank A.; Vincze, Imre E.; Wadhwa, Jagdish C.; and Zaman, Fasih Qamar uz.

Letters to the Editor

To the Editor:

I neglected to acknowledge the assistance provided by Dr. Harold Osher and the Maine Regional Medical Program in studying the patient with Adams-Stokes attacks due to paroxysmal ventricular tachycardia reported in December (Volume 63; 266, 1972). The portable EKG recorder used in this case was on loan from the Maine Regional Medical Program. Dr. Harold Osher kindly provided the scanning and review of the recorded tracing.

JOE R. WISE, JR., M.D.
1 Fern Street
Bangor, Maine 04401

To the Editor:

Lung Cancer Deaths Are Terrible — The Best Treatment Is Prevention

Death from lung cancer is a horrible death. As a physician, I have seen much of it and know that removal of one lung saves the patient in only a very small percentage of such cases. Frequently both lungs are affected and it is such a tragedy when an individual dies of a disease which *could have been prevented* by simply *not smoking*. It is true that Arthur Godfrey survived a lung removal but he had tears in his eyes as he was wheeled into surgery and would have given all the wealth of the Indies if he had never begun smoking. It is reported that he is smoking again. I am deeply distressed to hear that. If true, he like many others cannot stop a bad habit after it has fastened control over one's mind.

The tobacco advertisements show healthy, robot people opening cigarette packages, usually in Western cattle country, God's country, where the ads infer a rancher's day is not complete without the "relaxation" and "pleasure" of deeply inhaled death dealing tobacco smoke. The scene may shift to the ballroom or drawing room of a fine mansion where the cigarette is simply taken for granted for those who are "in."

But a lung cancer death is not like that; instead the patient often finds himself in great distress from uncontrollable coughing, the feeling of being "choked to death" and near death's door.

Those who felt they did not care suddenly do, but realize they can no longer be "busy" healthy people and tears or regrets won't wash away the awful truth.

I believe I know something about lung surgery, since I was one of six young full-time student physicians who worked with Evarts A. Graham, M.D., Professor of Surgery at Washington University Medical School, the pioneer who removed the first human lung in history. Dr. Graham saved many lives by this dramatic step but himself he could not save. He himself died of cancer of both lungs. At that time, only a few believed that there *was* a connection between smoking cigarettes and the terrible disease.

Like so many physicians, he had been a rather heavy smoker. Today most intelligent, strong willed and dedicated doctors have given up the dangerous habit altogether before it is too late to save their own lives and set an example for their patients, relatives and friends. They are ahead of all others for they know smoking kills through causing lung cancer, emphysema and heart disease.

Even before Dr. Graham's world renowned history-making operation by successfully removing a man's lung, I began to take a cigarette history on all patients I could locate who were diagnosed lung cancer. For while a chest patient in army hospitals, I saw that those diagnosed lung cancer and those who died of it were heavy smokers. I had this firm belief based on pt. histories just after our first Armistice Day in 1918, 30 years before the 1964 world-shaking 10 man report of the U.S. Surgeon General published everywhere saying, softly "Smoking may be hazardous" and giving much evidence from the accumulated data they studied that it was related to lung cancer.

In 1919, I occupied a bed in an army hospital with the diagnosis "pulmonary Tbc, moderately advanced both lungs." In a glass room next to mine was a captain who suffered much from coughing and "lung cancer." He was sure his lung cancer was due to smoking and he helped persuade me to believe that all lung cancer cases were "inveterate smokers." Later, I took smoking histories for 5 years in cancer hospitals and to myself if not the reading public or to the editors who threw my releases into the waste basket – proved that smoking causes lung cancer and I began saying what I still say, *Nobody can afford to smoke.*

During the thirties, for five years, I was daily inside skin and cancer hospitals and notified about every pt. diagnosed lung cancer. I then took cigarette histories on them and I became completely convinced that cigarette smoking and lung cancer and heart disease were definitely related. It was proof of my suspicions arrived at in the twenties when a long-time patient in chest wards of army hospitals that cancer of the lungs, at least statistically was directly related to smoking. I was saying "Nobody Can Afford to Smoke" therefore before the United States government had even taken a passing interest in tobacco except to indirectly raise and sell it. This was before there was other good statistical proof available. Of course, I already had known that cancer of tongue, lip, larynx and oral cavity was related to smoking of cigarettes, cigars and pipes, but in the January 14, 1938 issue of "The Church World" I first publicly announced my firm belief on the true cause of lung malignancy, and some heart conditions. To help people save their own lives, I have reiterated this conviction often in the interviewing years both publicly and privately.

The most successful way to control lung cancer and some heart diseases and an inexpensive and effective way is to simply *stop smoking.*

The fact that I have been saying that for more than thirty years with so little effect amazes me and I must confess I still often feel like a lamb bleating but not heard in a terrific North Dakota blizzard. It is a further mystery to me that now with every package of cigarettes placarded by U.S. Federal Law as *dangerous,* the sale of cigarettes and tobacco even now increases. Each day stupid boys and girls begin to smoke.

That my years of preaching against smoking has had so little effect is one of the disappointing experiences in my many years of trying to be a dedicated doctor and placing his patients' interest first.

ADRIAN H. SCOLTEN, M.D.
32 Deering Street
Portland, Maine 04101

Continued from Page 43

vasodilatation through a reduction in resting sympathetic coronary constrictor tone. The reduction in myocardial oxygen requirements and possibly increased supply has proven to be beneficial to patients utilizing electrical carotid stimulation to terminate attacks of angina pectoris.

Since the main function of the carotid sinus reflex is to regulate arterial pressure, carotid sinus nerve stimulation has been utilized in the therapy of the most common disorder of blood pressure, essential hypertension. Altered function of this reflex may contribute to sustained hypertension in this disorder. Persistent electrical carotid sinus stimulation has lowered blood pressure substantially for as long as 5 years. In some cases actual regression of eyeground changes have occurred. This technique, in association with antihypertensive drug therapy, is being used successfully for the treatment of moderate and severe hypertension.

Activation of the carotid sinus reflex is a useful diagnostic tool. Experiments in animals by Vatner and associates have demonstrated that the characteristics of the reflex change with altered states of consciousness, i.e., a differential pattern of autonomic outflow occurs in the conscious organism as opposed to during sleep or after general anesthesia. Baroreceptor function can be assessed in man by measurement of heart rate after administration of angiotensin intravenously. This drug raises arterial pressure by increasing peripheral resistance. The elevated arterial pressure then causes reflex slowing of heart rate. The relationship between the rise in arterial pressure and reflex slowing has been characterized in normal individuals and has been found to be altered in patients with hypertension and those with congestive heart failure. Diminished baroreceptor sensitivity has been demonstrated in both these disease states and may contribute to maintenance of essential hypertension.

Thus, the carotid sinus reflex, a cornerstone of cardiovascular physiology for half a century, is now being utilized in cardiovascular diagnosis and therapy. Electrical stimulation of the carotid sinus nerves is being employed to treat angina pectoris, recurrent supraventricular tachycardia, and essential hypertension. In addition, altered responsiveness of the baroreceptors in disease states may be valuable diagnostically and may provide further understanding of the pathophysiology of several circulatory disturbances.

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Frederic Henry Gerrish – Maine's Outstanding Figure in Academic and Clinical Medicine 1873-1920

Jean Alonzo Curran, M.D.

Too often an outstanding figure in the medical history of one generation is largely forgotten by those of us in the years to follow. Dr. Gerrish should be remembered in both New England and American medicine, along with such figures of distinction as Nathan Smith of Dartmouth, Daniel Drake of the Cincinnati Medical College, and Morton Prince of Tufts and Harvard Medical Schools.

Although I for one, have long appreciated the contributions made by the Gerrish Library to the Central Maine General Hospital, and to the early years of the Bingham Associates Fund program in that area, the source of the library's name escaped my attention.

So, Frederic H. Gerrish emerged as a person and a unique personality to me, for the first time in the fascinating biography of another famous Maine physician, Dr. Frederick J. Pritham, who died just recently after more than sixty-five years of devoted service to the people of the Greenville region. Fred Pritham was a student at Bowdoin's Medical School of Maine, under the control of Bowdoin College, (1900-1905). The story of those years contains references that whetted my interest: "He liked his courses and teachers, even Dr. Henry Gerrish, the dour and strict professor of anatomy, a fascinating study with all its casts and models, fresh and preserved specimens. . . ." ". . . Fred was not in awe of Gerrish."[1-p.34]

"A farm boy had an advantage in Gerrish's class, which encouraged the dissection of lower animals before coming to college."

When Dr. Pritham graduated, he was asked to provide references by the formidable Dr. Hiram Hunt of Greenville, who had been president of the Maine Medical Association the previous year. Fred mentioned Dr. E. J. Gray of Freeport, his sponsor and family doctor, and Professor Gerrish. "He was probably the only one in the class who would have dared to use the stern professor as a reference."[1-p.44]

That Gerrish was a scholarly contributor to the advancement of anatomical science and education is revealed by an impressive 917 page text book, with 950 fine engravings in black and color, of which he was the editor, as Professor of Anatomy at Bowdoin.[2] Although such recognized authorities as Arthur Dean Bevan of Rush Medical College, George David Stewart of the University and Bellevue Hospital Medical College, and other professors at Texas, Michigan and Cornell universities were contributors, Gerrish himself was the author of fifteen sections, totalling 419 pages of almost half of this valuable guide to student and graduate study.*

The next encounter with this outstanding professor of anatomy's story was a startling surprise – a communication from H. Edward MacMahon, M.D., Professor of Pathology, Emeritus, Tufts University Medical School and Director of Pathology, Tufts-New England Medical Center Hospital, and long associated with the late Joseph H. Pratt, M.D., and with the Bingham Associates Fund as a consultant in Maine. In a recent article in the Tufts Medical Alumni Bulletin,[3] Dr. MacMahon recounts verbatim an extraordinary service rendered to a patient by Dr. Gerrish as a psychotherapeutic practitioner (only one of the many fields of medicine to which this amazing man made significant contributions), as told to him by Dr. Pratt a short time before he died.

The story involves Dr. George Gehring of Bethel, Maine, who had retired from a surgical practice in Cleve-

*I am indebted to Reginald T. Lombard, M.D., of 793 Maine Street, South Portland for the generous loan of this fine volume.

land because of neurasthenic problems. According to Dr. Pratt, "He had married Mrs. Farnsworth, a well-to-do widow, who had one child, George, born in 1880. . . . Mrs. Gehring was also a psychoneurotic individual."[3]

After being treated unsuccessfully by a number of doctors, including the great Weir Mitchell, it was decided to seek the aid of Dr. Jacob M. DaCosta, Professor of Theory and Practice of Medicine, Jefferson Medical College. "On her way she became so ill in Portland that she was taken from the train and local aid sought in this emergency," in the person of Portland's outstanding physician, Dr. Gerrish, who ". . . was so successful in his psychotherapy that Mrs. Gehring refused to go on to Philadelphia and remained in Portland for several weeks, staying at the hotel and being treated by Dr. Gerrish. Dr. Gehring, who was in much the same state as his wife, that he wanted the same treatment . . ." and ". . . got so much out of his treatment and his wife was so much better, that he returned to Bethel a convert, and with missionary zeal set out to convince the local doctors in and about Bethel of this new form of treatment."[3]

According to Garland, "The Gehrings enjoyed travelling, and on one of the frequent European trips they met Dr. H. Bernheim, a French psychiatrist who had pioneered in treatment for the emotionally disturbed,"[4] and the Bethel doctor became enthusiastic about Bernheim's methods. At the same time he was much impressed by the techniques of Dr. Gerrish who practiced autosuggestion, and was strongly influenced by reading a treatise on "Suggestive Therapeutics." He was so convinced of the value of such treatments in the emotionally disturbed, that he resumed practice in Bethel in 1895, and was so successful in rehabilitating emotionally debilitated cases that he attracted patients from far and wide. Among them was William Bingham, a wealthy young bachelor from Cleveland. Out of his gratitude for the helpfulness of not only Dr. Gehring, but Drs. Pratt and Samuel Proger, the concept of the Bingham Associates Fund program was born – of such long term importance to all health activities in Maine. Hence, in this development, Dr. Gerrish's original inspiration played a vital part!

Actually Gerrish's rise to eminence as a psychiatrist was but a natural outgrowth of his breadth of exploratory interests in a wide range of the fundamental sciences, clinical medicine and public health.

After his thorough educational preparation at Bowdoin College; A.B. 1866, and A.M., M.D. 1869, he began his academic career at his Alma Mater as lecturer in therapeutics, materia medica and physiology in 1873-74. Because of the short school years at that time, he was able to serve also for one term as professor at the University of Michigan, 1874-75. For the ensuing ten years, his widening interest in community aspects of medicine was evidenced by his serving as Lecturer in Public Health, while continuing his career as Professor of Materia Medica and Therapeutics at Bowdoin until 1904, and then Professor of Surgery 1904-1911. Even in the retirement years he lectured on Medical Ethics from 1911 until his death in 1920!

According to information supplied by Philip K. Reiman, Executive Director of the Maine Medical Center, research into the Annual Reports of the then Maine General Hospital, Dr. Gerrish began his responsibilities there in 1869 as Pathologist; Secretary of the Medical Staff, 1871-1879; Visiting Surgeon 1879-1887; Attending Surgeon until 1893, and finally a member of the Consulting Staff.

Although it was Dr. Nathan Smith, founder of Dartmouth Medical School, who taught practically all of the original courses there in 1798, to whom Oliver Wendell Holmes referred: as occupying ". . . a whole settee of professorships . . .," this designation could have been equally applied to Frederic H. Gerrish from 1873 to 1920 at Bowdoin!

But he was not content to concentrate his range of interests entirely to the local scene of academic and clinical medicine. As early as 1885-89, his standing was recognized by election as President of the State Board of Health, and concurrently President of the American Academy of Medicine, 1887, followed by the presidency of the Maine Medical Association, 1901-1902, and American Therapeutic Association, 1908-1909. There were fellowships of the American Surgical Association, American College of Surgeons, and memberships of the Societé Internationale de Chirurgie and New England Surgical Society, and even on the Advisory Council on Simplified Spelling!

Dr. Gerrish was one of the pioneers in Maine in applying microscopic studies to the advancement of medical science. According to his close friend, Dr. W. E. Tobie of Portland, his first presentation before the Maine Medical Association in 1871 was entitled, "The Microscope in Medicine."[5]

The youthful Gerrish with his wide-ranging absorption in all fields of medical science must have been deeply impressed by Virchow's concept of the cellular nature of all life processes, both physiological and pathological, as revealed by his subsequent life work. Also, like Virchow, who had begun his career as an anatomist, he developed concerns for factors adverse to health in the environment, became versed in vital statistics, and presented a report to the Maine Medical Association on the findings of a survey of the distribution of infectious diseases, and of sewage disposal and the water supply of the City of Portland in 1877.[6] The existing Board of Health was criticized for not having a member with medical training. A year later he challenged the medical profession to meet its responsibilities with regard to prostitution and its allied vices as menaces to public health.[7]

Recognition of the role of bacterial infection in surgical operations made Gerrish an early convert to "Listerism." On September 11, 1880, he reported to the Portland Clinical Society on a series of patients treated by the Lister procedure, shortly after being appointed Visiting Surgeon at the Maine General Hospital. After only

four months experience, there was a comprehensive report of complete antiseptic technique; including efforts to sterilize the air of the operating room, instruments, sponges, sutures and hands of the operators: all 'carbolized,' and then the employment of the complete Lister dressings. In a series of breast amputations, the results were a dramatic contrast to a series of previous operations with following suppuration. Gerrish's patients remained clean of infection! The following year he published a translation from the French literature of a work on antiseptic surgery by Lucas Championiere.[8]

Gerrish as a Psychotherapist:

That Gerrish should have acquired an interest in mental health is not surprising, for it was a part of the Bowdoin tradition. As early as 1843, William Sweetser, the Bowdoin Professor of the Theory and Practice of Physic published a book on Mental Hygiene. Gerrish's recognition of the 'functional' as well as the 'organic' ailments among the large number of patients in his private practice was undoubtedly stimulated by the pioneering contributions of such figures in the field as Charcot, Janet, Freud and Morton Prince of Boston with whom he shared an interest in the therapeutic use of hypnosis. In 1891, he became a member of The Society for Psychical Research, as well as The American Anthropometric Society. By 1892, he was prepared to take a definite stand on the remedial uses of hypnotism, and presented his findings at the Annual Meeting of the Maine Medical Association.[9]

In Gerrish's Foreword to his publication, it was explained that in addition to the record in the Society's Transactions, he was putting the presentation into printed form as a pamphlet to reach a wider circle of friends. Obviously because of the dangers of sensationalism, he carefully explained that this publication was in no way motivated to ". . . enlarge his practice in the neurological line, his special taste lying in an altogether different direction." He added the significant comment, "Having found himself apparently alone among his immediate associates in his appreciation of the therapeutic value of hypnotism, he has endeavored in the past year to interest his fellow practitioners in this little-understood remedy."[9]

He went on to explain that ". . . the phenomena of what we call hypnotism have been recognized, under one or another name, for thousands of years – in the present century quite commonly as animal magnetism, odylic force, mesmerism, electro-biology, neurhypnotism and braidism. . . ." ". . . the key to some of the most perplexing problems in psychology will possibly be found in this mysterious agency." "Hypnotism is an induced condition in which the mind is peculiarly susceptible to suggestion."

In a footnote he elucidated further, "Suggestion is only the artifice employed to induce the phenomenon. The real psychic mechanism of the process is still undetermined."

Gerrish was speaking from very extensive experience: "For more than a year past I have used hypnotism almost daily in my practice," (with) ". . . personal observation, which now covers about fifteen hundred applications."

In view of speculation in 1972 as to whether hypnotism may be a factor in the reported success of acupuncture anesthesia in China, Gerrish's observations in this connection are of interest:

"For the production of surgical anesthesia, hypnotism is not generally available, because so small a percentage of people can be put into the somnambulic stage, in which operations of any considerable magnitude can be performed without causing suffering. But blunting of sensibility sufficiently pronounced to prevent pain in minor procedures, like excavation and filling carious cavities in teeth, can be accomplished with the patient in the lighter degrees of the hypnotic state, if suggestion of freedom from pain be repeated incessantly during the operation. This I have not seen mentioned in any of the works on hypnotism and I believe it to be a valuable discovery, the credit of which should be accorded to our fellow member Dr. Thomas Fillebrown, of Portland, Professor of Operative Dentistry in Harvard University." (This was followed by a series of case reports.[9])

His interest continued throughout his career, for he participated in the Annual Meeting of the American Therapeutic Society in 1908, and contributed to a collection of papers published in book form entitled, "Psychotherapeutics." At the meeting it was recorded that, "Its President, Dr. F. H. Gerrish, Professor Emeritus of Surgery at Bowdoin College, arranged the program and obtained the contributions from Morton Prince, J. J. Putnam, Boris Sidis and other well-known figures. Dr. Gerrish's own chapter dealt with hypnotic suggestion and E. W. Taylor wrote on, "Simple Explanation and Re-Education as a Therapeutic Method."

In March 1910, Gerrish prepared an article for a symposium on Psychotherapeutics in which he strongly supported the value of hypnosis, saying,

"There are a multitude of errors concerning hypnotism, which are almost as prevalent amongst physicians as in the nonmedical population. . . ."

"It is commonly supposed that a person must lose consciousness in the process. In most cases, the patient retains consciousness perfectly. . . . Brilliant cures are sometimes achieved with patients who are hardly made drowsy by being hypnotized."

"The great majority of people can be hypnotized to an extent quite sufficient to make them susceptible to remedial suggestion." (Dr. Gerrish denied that the hypnotist had to possess) . . ., "peculiar and rare natural endowments. He who has a strong and healthy physical organism, a powerful mind, and perhaps best of all, an engaging presence, has the elements that make for success, in any affairs where man meet man."[10]

One can only comment that Dr. Frederic Henry Gerrish himself must have been endowed with these qualities to an superlative degree! When he died on September 8, 1920, the graduates of Bowdoin Medical School, outnumbered the practitioners from other sources in the state, and they joined in revering his memory as the greatest faculty figure of their era. Hence, it was especial-

ly appropriate that they or their representatives and other colleagues; 125 doctors, their wives and distinguished guests should meet on May 26, 1937 to dedicate the Frederic Henry Gerrish Memorial Library at the Central Maine General Hospital, Lewiston, at an afternoon program and a banquet at the DeWitt Clinton Hotel.

This had been made possible by the special efforts of Dr. Julius Gottlieb, Director of Pathology at the Hospital, when he enlisted the active interest of Dr. Joseph H. Pratt and Dr. Samuel Proger, to secure the grant from the Bingham Associates Fund to make the new Library possible.

Dr. Paul Dudley White honored the occasion by conducting the afternoon clinical program. At the banquet, President Clifton D. Gray of Bates College gave the invocation, and Dr. Frederick T. Hill of Thayer Hospital provided introductory remarks as President of the Maine Medical Association.

Dr. W. E. Tobie of Portland was the dinner speaker. After reviewing the highlights of the Gerrish career, he explained that his acquaintance with Dr. Gerrish dated back to 1887, but was especially intimate from 1897; as student, assistant and associate in both anatomy and surgery, with the distinction of succeeding to those professorships. In testimony to the austerity and sternness of manner (as so vividly recalled by Fred Pritham), Tobie commented that, "Dr. Gerrish's ability commanded universal respect. He had many admirers but few close friends. . . . Dr. Gerrish made a distinct impression on medicine in the State of Maine. No one in the history of Bowdoin Medical School and the Maine Medical Association approached him in ability and in the influence he wielded. His influence was always for good and he stood for what he believed to be the right."[11]

Other tributes came from several other former students of Dr. Gerrish; Dr. E. V. Call and Dr. C. B. Sylvester of Portland, Dr. G. H. Rand of Livermore Falls, and Dr. A. W. Plummer of Lisbon. Dr. Pratt spoke as representative of the "B.A.F.," followed by Mr. Samuel Stewart the Director of the "C.M.G.H.," and Dr. R. J. Wiseman of the Ste. Marie's General Hospital.

The auspicious occasion was concluded with a technical paper by Dr. White on "Doctors and Books."

Acknowledgement

I am deeply indebted to Mr. Dana S. Thompson, Executive Director, Central Maine General Hospital, for his provision of extensive reference material about Dr. Gerrish, and a list of Gerrish Memorabilia, preserved at the Hospital Library, including Dr. Gerrish's desk, writing materials, a loving cup presented by the Bowdoin Class of 1907 and a variety of personal articles, preserved in a display case.

Mr. Phillip K. Reiman was most helpful in supplying helpful information about Dr. Gerrish's great career at his hospital center, including the loan by Dr. Lombard of South Portland of the Gerrish text book of anatomy.

Special appreciation must be expressed to Mr. Richard B. Reed, Librarian Special Collections, Bowdoin College Library for a copy of; "Frederico Henrico Gerrish('s) Dissertatio de Sectione Caesarea," MDCCCLXIX –. This is Gerrish's graduation thesis preserved in the Bowdoin College Library (as are all those receiving the M.D. degree over the more than a century of the Medical College's existence). In his exposition, Gerrish offered convincing evidence for the advantages of Caesarian operation over difficult forceps deliveries, even in those pre-antiseptic years. During his years in private practice, Dr. Gerrish included obstetrics in his wide-ranging interests.

Finally, Mr. Arthur Monke, Librarian of the Bowdoin College Library was most helpful, from the beginning of the search for Gerrish material, including letters of Professor Sweetzer to Parker Cleaveland, the Gerrish presentation before the Maine Medical Association in 1892, entitled "Remedial Uses of Hypnotism."

References

1. Dorothy Clarke Wilson: "The Big-Little World of Doc Pritham." McGraw-Hill Book Company – 1971. pp 34, 35, 44.
2. Text-Book Of Anatomy, By American Authors, edited by Frederick Henry Gerrish, M.D., Lea Brothers & Co., Philadelphia and New York. 1899. 917 pages.
3. H. Edward MacMahon, M.D.: "Tufts University School of Medicine In Retrospect – 1929 – 1071." Tufts Medical Alumni Bulletin, June 1971, Volume 30, No. 2. "The Bingham Associates Program, (1930-1950), pp 16-20.
4. Joseph E. Garland, Jr.: "An Experiment In Medicine." The Riverside Press, Cambridge, Massachusetts, 1960. pp 2-4.
5. W. E. Tobie: Address at the Dedication of the Gerrish Memorial Library, May 26, 1937.
6. F. H. Gerrish, M.D.: "The Sanitary Condition of Portland," Report to the Maine Medical Association, June 14, 1877, reprinted from the Proceedings of the Association.
7. F. H. Gerrish, M.D.: Oration at the Annual Meeting of the Maine Medical Association, June 12, 1878.
8. Lucas-Championiere – "Antiseptic Surgery," translated by Frederic H. Gerrish, 1881 . 8°, Portland, Maine (copy in the Bowdoin Library).
9. Frederic Henry Gerrish, A.M., M.D.: "The Remedial Uses of Hypnotism." Essay read at the Annual Meeting of the Maine Medical Association, June 9, 1892. William Marks, Printer, 1892.
10. Professor F. H. Gerrish: "M.A.B." (Mainly About Books), from, "Psychotherapeutics: A Symposium." Article – Vol. III, No. 9, March 1910, p. 175 (Bowdoin Library).
11. W. E. Tobie, M.D.: Address at the Frederic Henry Gerrish Library Memorial dinner, May 26, 1937. Reported in The Lewiston Daily Sun, May 27, 1937.

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Editorial

T. V. Medicine

I feel it is appropriate at this time to point out some of the health hazards of buffered and effervescent analgesics which are cleverly advertised widely in the television media. As I am a gastroenterologist and see many of the untoward effects of such preparations, I deem it necessary to warn physicians to caution their patients against believing literally that aspirin is a treatment for indigestion or gas, or will cure the after effects from overindulgence in food and beverage. A particular offender is the manufacturer of an effervescent aspirin of "I thought I was going to die" fame who might serve the public better by omitting the aspirin from the preparation and just giving the antacid.

It should be pointed out that the amount of buffering capacity in any of such preparation is insufficient to substantially neutralize stomach contents for any significant length of time. The apparent prime purpose of antacids in these preparations, especially those containing carbonates, is to rapidly disperse the ingested tablets so that the aspirin will be available for absorption. It must also be noted that aspirin in its ionized form, the form that exists in alkaline media, is actually less absorbable by stomach mucosa than is the unionized form. Because it is less absorbable, it is less irritating to the stomach. Therefore, most of ionized aspirin is absorbed in the duodenum or beyond. Nevertheless, aspirin finally absorbed in the blood stream is systemically irritating to the gastric mucosa, and by the time aspirin reaches peak concentrations in the blood, the trivial amount of buffering capacity of the original preparation has long been overwhelmed by stomach acid. The combination of normal or high acid in the stomach with aspirin sets the stage for further irritation of an already "too full tummy."

Patients who happen to be hypersecretors of acid, the most likely patients to have dyspepsia in the first place, would be the most likely to have adverse effects from ingested aspirin. Indeed, it is often ulcer patients who take aspirin, aspirin containing compounds, and buffered or effervescent aspirin compounds for the relief of abdominal pain due to an ulcer! Nothing could be worse for these patients and often bleeding or obstruction is precipitated.

In short, present ingenious advertising has captured the public and led to the gross overuse of aspirin for primary stomach complaints. It would be better for all concerned to take mere bicarbonate of soda in water – a product which is probably just as ubiquitous in the home as aspirin.

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Dates For Your 1973 Calendar . . .

Interim Meeting, M.M.A. House of Delegates — April 8

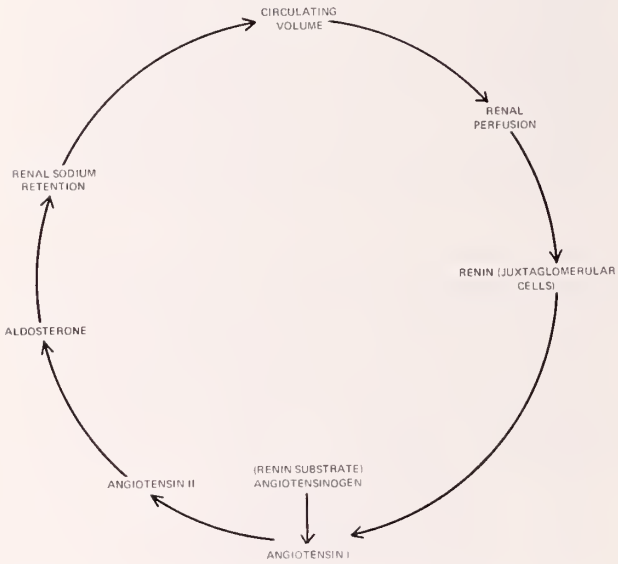
M.M.A. Annual Session — June 16-19

Accelerated Hypertension Secondary to Oral Contraceptives

Stephen A. Sokol, M.D.*

In 1955, aldosterone was first conclusively linked to hypertension by Doctor Jerome Conn. During the following few years, the elucidation of the renin-angiotension-aldosterone system took place.

Diagram #1



As can be seen from the accompanying diagram, renin is secreted by the juxtaglomerular cells, which surround the afferent arterioles of the renal cortical glomeruli, in response to physiologic stimuli which have volume depletion as a common factor. Renin then acts on the renin substrate, angiotensinogin, an alpha – 2 globulin, manufactured by the liver. The product formed is a decapeptide called angiotensin I. This is further split by a "converting enzyme" in the plasma to angiotensin II. Angiotensin II, by itself, is one of the most powerful vasopressors (hypertensive) agents known and, in addition, stimulates the adrenal cortex to secrete aldosterone. It has been known since 1952 that estrogens have an effect on the renin-angiotensin system but it was not until 1966, about 10 years after the introduction of oral contraceptives (estrogenic and progestational compounds in combination), that the association between them and hypertension became appreciated.

Recently a patient with severe accelerated hypertension secondary to "the pill" has been studied and can serve as a focus for a discussion of a potentially curable form of hypertension that is often overlooked.

*Central Maine General Hospital, Lewiston, Maine 04240.

Case History

Mrs. R. C., a 29-year-old lady, was admitted to the hospital for evaluation of severe headaches and hypertension. She first began taking oral contraceptive agents in 1964 and discontinued them in 1965. In 1966, she was restarted on "Ovulen®" (contains 0.10 mg. estradiol) and in April 1971 she was switched to "Ovral®" (contains 0.05 mg. estradiol). She remained well until six months prior to admission when she noted an increase in frequency and severity of her "tension" headaches. Formerly occurring once or twice monthly they now recurred biweekly, lasting from one to three days. One week prior to admission she was seen by her family physician who noted a blood pressure of 204/122 and referred her for evaluation. Her blood pressure in 1964 was noted to be 110/80 and in 1969, 140/90. She had a past history of mild hypertension with her pregnancies as well as minimal edema but never toxemia. She had symptoms on one occasion of a urinary tract infection. This had been treated successfully. In the family only her father had high blood pressure, evidently well controlled on medication.

Physical examination on admission showed a healthy appearing woman complaining bitterly of a severe headache. Her blood pressure was 240/150 bilaterally. Systolic blood pressure in the leg was 230, pulse 100 and regular, respirations 16. There were Grade II hypertensive changes in the fundi with AV nicking but no papilledema, hemorrhages, or exudates. The heart was slightly enlarged to the left. There was an S4 gallop and a markedly increased aortic sound. There were no other pertinent physical findings.

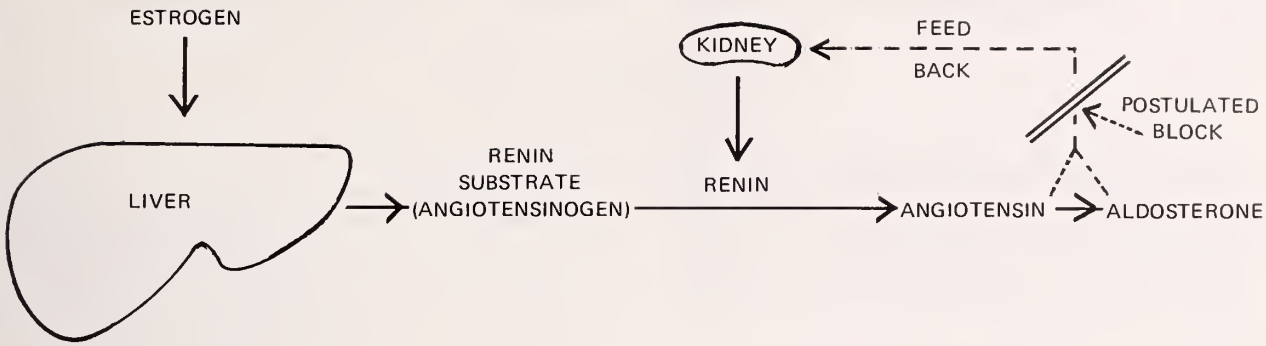
The CBC was normal as was the SMA 12/60. BUN was 16; Creatinine, 1.3; T3, 21%; and T4, 8.8 micrograms (consistent with changes secondary to oral contraceptive agents). Urinalysis was unremarkable except for pyuria. Urine cultures grew out repeatedly over a 100 thousand colonies of E coli. KUB showed a left kidney that was greater than the right kidney. Because of the difference in renal size, renal arteriography was performed. There were no abnormalities of the aorta or either renal artery. Nephrogram showed the left kidney approximately 4 cm. greater than the right with evidence of chronic pyelonephritis in the right kidney.

Course: The patient's oral contraceptive agent was discontinued. She was started on HydroDIURIL®, 50 mg. twice daily, and discharged from the hospital. When seen in follow-up one week later, her blood pressure had dropped to 110 to 120 / 70 to 80 and her headaches had diminished markedly. The HydroDIURIL was discontinued. On follow-up examination one month later, her blood pressure was averaging 170/110 and the HydroDIURIL was restarted. Later follow-up showed her blood pressure in the range of 110 to 130 / 80 to 90 and her most recent examination showed that her blood pressure was 110/80. At this point her HydroDIURIL was once again discontinued.

Discussion

The effects of both estrogens and progesterones on the renin-aldosterone system were investigated by Cram and Harris in 1968.[6] They conclusively showed that the effect of progesterone compounds was to decrease aldosterone excretion rate and that they (progesterone compounds) had no effect on the plasma renin activity (renin-angiotension system). Additionally, they and several other investigators[3–6, 12,16] showed that estrogens increase the

Diagram #2



amount of renin substrate markedly. The increased substrate was associated with an increased generation of angiotensin (plasma renin activity). Moreover, renin substrate has been found to be markedly elevated in normal pregnancy.[3]

Skinner[5] found that normotensive females on oral contraceptive agents had a decrease in plasma renin concentration suggesting a decreased secretion by the kidney. It is known that both angiotensin and aldosterone decrease renin secretion by the kidney. He suggested that women who become hypertensive on "the pill" might be unable to suppress renin secretion normally. This was further studied by Saruta[14] who found, in a prospective study, that 11 out of 62 women became hypertensive on "the pill." Of these, 8 developed abnormally elevated renin concentrations confirming a loss of feedback control.

The reason for the loss of feedback inhibition is not known. However, recently in a letter, Ndeti[16] noted that a high salt intake decreased blood pressure in his patients made hypertensive by oral contraceptive agents. It is known that salt infusion (volume expansion) will decrease renin secretion. This suggests that the feedback control in these hypertensive women becomes insensitive to angiotensin or aldosterone or both for unknown reasons.

The magnitude of the problem is not known. In two retrospective studies,[11,13] no significant hypertension was discovered. And, in the only two prospective studies[14,15] published, 15-18% of women studied became hypertensive. The difference may be related to the relatively short period of study in the retrospective analysis –3 months in one and 1 year in the other. It has been noted[7] that hypertension developed anywhere from 3 to 36 months and that the average interval was approximately 1 year. It is possible that further retrospective analysis over a longer period would have shown the development of significant hypertension.

Hypertension should be considered a contraindication to birth control pills. Patients on oral contraceptive agents should be evaluated frequently for the development of hypertension and if hypertension is observed these drugs must be discontinued.

As a corollary, although I was unable to find any reports in the literature, it must be assumed that post-menopausal women on replacement estrogen therapy run the same risks. Hypertension, likewise, should be a contraindication to hormonal replacement therapy and this therapy should be discontinued if elevated blood pressure occurs.

Addendum

A further search revealed that one group* studying this problem found five patients who became hypertensive while taking conjugated estrogens (Premarin®). Two of these were investigated and discovered to have hyporesponsive plasma renin activity. A direct mineralocorticoid-like effect with secondary suppression has been postulated.

*Craine, M. G., Harris, J. J. and Windsor, W., III: "Hypertension, Oral Contraceptive Agents, and Conjugated Estrogens." Annals of Internal Medicine; 74: 13, 1971.

References

1. Helmer, O. M. and R. S. Griffith: "The Effect of the Administration of Estrogens on the Renin substrate (hypertensionogen) Content of Rat Plasma." Endocrinology; 51; 421, 1952.
2. Wood, J. Edwin: "The Cardiovascular Effects of Oral Contraceptives." Modern Concepts of Cardiovascular Disease; XLI; #8, August 1972.
3. Helmer, Oscar M., Judson, Walter E.: "Influence of High Renin Substrate Levels on Renin-Angiotensin System in Pregnancy." American Journal of Obstetrics and Gynecology; 99, #1; September 1, 1967.
4. Katz, Fred H., Kapas, Attallah: "The Effects of Estradiol on Plasma-Levels of Cortisol and Thyroid Hormone-Binding Globulins and on Aldosterone and Cortisol Secretion Rates in Man." The Journal of Clinical Investigation; 46; #11, 1967.
5. Skinner, S. L., Lumbers, Eugenie R. and Symonds, E. M.: "Alternation by Oral Contraceptives of Normal Menstrual Changes in Plasma Renin Activity, Concentration and Substrate." Clinical Science; 36; 67-76, 1969.
6. Cram, Milton G., Harris, John J.: "Plasma Renin Activity and Aldosterone Excretion Rate in Normal Subjects. I. Effect of Ethinyl Estradiol and Medroxyprogesterone Acetate." Journal of Endocrinology; 29; 500, 1969.
7. Weinberger, Myron H., Collins, R., Dennis, Dowdy, Anne J., Nokes, George W. and Luetscher, John A.: "Hypertension Induced by Oral Contraceptives Containing Estrogen and Gestagen." Annals of Internal Medicine; 71; 891-902, 1969.
8. Walters, W. A. W., Lim, Y. L.: "Cardiovascular Dynamics in Women Receiving Oral Contraceptive Therapy." Lancet; 879-881, 1969.

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Procainamide and Lupus Erythematosus

Stephen A. Sokol, M.D.*

In 1948, Hargraves and his colleagues at the Mayo Clinic described the Lupus Erythematosus (LE) Cell phenomenon. Within three years, reports began appearing linking various drugs with the production of this syndrome. Approximately thirty drugs have been implicated and most recently[14] quinidine has become associated.

Table 1

Drugs Implicated in Inducing Lupus Erythematosus

Aminosalicylic acid	Para-aminosalicylic acid (PAS)
Chlortetracycline (Aureomycin®)	Penicillin
Digitalis (long term)	Phenobarbital (long term)
Diphenylhydantoin (Dilantin®)	Phenylbutazone (Butazolidin®)
Ethosuximide (Zarontin®)	Primidone (Mysoline®)
Gold compounds (long term)	Procainamide (Pronestyl®)
Griseofulvin	Propylthiouracil
Guanoxan	Reserpine (long term)
Hydralazine (Apresoline®)	Streptomycin
Isoniazid (Nydrazid®)	Sulfadimethoxin
Isoquinazepon	Sulfamethoxypyridazine (Kynex®)
Mephenytoin (Mesantoin®)	Sulfonamides (long acting)
Methyldopa (Aldomet®)	Tetracycline
Methylthiouracil	Thiazides (long term)
Oral contraceptives (mestranol)	Trimethadione (Tridione®)

Of these, procainamide (Pronestyl®) has been increasingly recognized as the most potent stimulator of drug-induced serological changes and the lupus erythematosus syndrome. One of my cardiac patients developed a hemolytic anemia secondary to procainamide, an uncommon but not rare complication and part of this syndrome.

Case Report

R. F., a 52-year-old man, developed symptoms of coronary artery disease in 1970. Shortly thereafter he was hospitalized for the first of three myocardial infarctions. In June of 1971, he underwent saphenous vein bypass grafting because of severe triple vessel disease. He remained well until October 1971, when he developed mild gradually progressive exertional dyspnea. In November, right upper lobe pneumonia developed which gradually cleared. Early in February, orthopnea and nocturnal dyspnea occurred and he was admitted to the hospital in congestive heart failure. He was treated successfully with digitalization, low salt diet and Lasix®. Because of frequent premature ventricular contractions, quinidine was started. However, this was discontinued because of nausea, vomiting and diarrhea. Procainamide, 250 mg. four times a day, was substituted. In March, he was readmitted because of paroxysmal atrial fibrillation. He converted on oral propanolol and was maintained on his procainamide. Later in the month, because of an increase in frequency of ventricular premature contractions, the drug was increased to 500 mg. every six hours. His condition remained stable until late in May when he began developing increasing abdominal discomfort, shortness of breath and vomiting. Because of these symptoms he was admitted to the hospital early in June.

Physical exam disclosed a pale, tachypneic man, complaining of shortness of breath. His blood pressure was 110/70, pulse 100, respirations 20. He was afebrile. Pertinent physical findings included grade I arteriolar changes in the fundi, flat neck veins and diffuse inspiratory and expiratory rales in the chest posteriorly. The heart was enlarged two centimeters to the left of the midclavicular line. There was an S4 gallop and a grade III blowing pansystolic murmur best heard along the left sternal border with radiation toward the apex. The liver was one finger-breadth enlarged and the spleen was not palpable.

His CBC initially showed 10.2 grams of hemoglobin with a hematocrit of 31%, falling in two days to 8.9 grams and 26.7% (it previously had been in the range of 13 grams and 37%). His white count was 5,800 with a slight shift to the left and the reticulocyte count was 3.5%. Cells were normochromic and normocytic. Serial enzymes were normal except for an elevated LDH. The BUN was 30.5 falling to 23 two days later. Coombs test, direct and indirect, was positive. Attempted cross-match revealed autoagglutins. Plasma hemoglobin was 223 mg.%; bilirubin, 0.6 mg.%; haptaglobin, 175 mg.%; urine for hemocyterin was negative. Electrocardiogram showed a left bundle branch block; a portable chest x-ray showed minimal congestive changes in both lung fields and lung scan was unremarkable. Antinuclear antibody titre was 1/4000 homogenous and 1/512 peripheral nodular. C3 (complement level) was 129 mg.% (normal); C4, 5 mg.% (low).

Course: In addition to his cardiac diagnoses, a diagnosis of hemolytic anemia secondary to procainamide was made. This drug was stopped and quinidine was substituted. When stabilized, he was transferred to Boston for repeat cardiac catheterization. While there his hematocrit rose to 33.4%; his hemoglobin to 11.1 grams and his reticulocytes to 5%. His Coombs test remained positive. His hematocrit and hemoglobin one month later remained in this range and a repeat antinuclear antibody titre was planned but the patient died suddenly in mid-August.

This complication prompted a review of all my patients receiving procainamide as an antiarrhythmic drug.

The antinuclear antibody (ANA) test is the most reliable method of following patients receiving this drug. No patient with drug-induced lupus erythematosus and a negative ANA titre has been reported. Although it has been shown[3,10] that the height of the ANA titre does not correlate with the symptoms, it is accepted that development of a significant titre is an indication for stopping the drug. Two prospective studies[4,5] used the ANA test or variations of it to follow the development of serologic changes. In one,[4] seventeen out of twenty-two patients (77%) developed positive ANAs up to 1/64. In the other,[5] eight out of sixteen patients developed positive ANAs within nine months. The earliest appeared at nine weeks and most by four months. Two of these patients developed symptoms. In a retrospective review, Fakhro et al[3] described fifteen out of less than fifty patients who developed features of this syndrome. They distinguished three groups:

1. Asymptomatic patients with only serologic changes (four out of fifteen).

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TABLE 2

Case #	Age	Sex	Dose & Duration	ANA	Symptoms
1+	44	M	1 gram/day for 2 days	—	Drug fever
2+	61	M	1 gram/day for 6 months	1/1024	Arthralgias for 2 weeks
3+	67	F	1 gram/day for 1 week	1/2	Drug fever
4	65	M	1 gram/day for 1 year	1/4	No symptoms
5+	53	M	1 gram/day for 2 months	1/256	No symptoms
6	65	M	2 grams/day for 6 months	1/64 at 2 months 1/16 at 6 months	No symptoms
7*	75	F	1 gram/day for 18 months	1/64 at 6 months 1/64 at 1 year	No symptoms
8	61	M	1.25 grams/day for 6 weeks	1/32	No symptoms
9	63	M	1 gram/day for 10 months	1/4	No symptoms
10	76	M	1 gram/day for 7 months	1/8	No symptoms
11+	46	M	1 gram/day for 3 months	1/128	No symptoms
12	39	M	1.5 grams/day for 11 months	1/2	No symptoms
13	65	M	1 gram/day for 10 months	1/4	No symptoms
14**	66	M	1 gram/day for 6 months	1/2	No symptoms
15	72	M	1 gram/day for 8 months	1/2	No symptoms
16+ case report	52	M	2 grams/day for 4 months	1/4000	Hemolytic anemia

*Granddaughter has rheumatic heart disease
**Patient has rheumatic heart disease
+Drug Stopped

2. Symptomatic patient with serologic changes and only musculoskeletal complaints (five out of fifteen).
3. Those with multisystem involvement (six out of fifteen).

In the second review,[6] Russell and Ziff described eighteen patients with the drug-induced lupus syndrome. All eighteen had positive ANAs. Of twenty-three asymptomatic patients on procainamide, seventeen (70%) had positive ANAs ranging from 1/4 to 1/64.

There is no question that procainamide will induce a positive ANA in many patients for poorly comprehended reasons. Russell and Ziff[6] found a naturally occurring antibody to the drug in approximately one-third of the subjects they studied. This varied from 0% in neonates to 30% for normal adults to 50% for people taking the drug. The antibody was in the IgM Class where most naturally occurring antibodies are. Moreover this antibody was found in only 6% of patients with drug-induced lupus. They postulated that the antibody was undergoing absorption from the serum, either in reaction with tissue components or as a circulating antigen-antibody complex.

The procainamide induced lupus syndrome differs from the natural, not only in the age of onset (older patients as they are most often exposed to the drug), sex (no female prediliction) but also in symptomatology. The earliest presenting complaint and best warning sign is arthralgias. There is greater pleuropulmonary involvement and no renal disease. Stopping the drug alone or in conjunction with the use of steroids, usually produces a rapid reversal of the disease, although an occasional patient[3] has long-lasting complaints, usually arthritis. Permanent sequelae can ensue,[7] but probably only if early signs and symptoms are ignored.

The disease is neither dose nor time related. One extensive review by Sheldon and Williams[10] pointed out that the dose of procainamide varied from 0.5 to 6 grams and that the duration of therapy before symptoms appeared ranged from two weeks to six years.

There has been controversy in the literature regarding a lupus-diathesis in patients using these drugs. In one study[9] of four children with lupus induced by anticonvulsants, eighteen out of forty-four (41%) asymptomatic relatives had increased gammaglobulins, positive rheumatoid histories or positive rheumatoid serologic factors. Several authors[7,8,10,11] feel that this diathesis definitely exists. Others,[4,5,12] disagree. Certainly the two most cogent arguments against this diasthesis are the appearance of serologic changes in at least 50% of patients using procainamide, and the finding of naturally occurring antibodies to this drug in 30% of normal adults[6] not exposed to it.

My personal experience with sixteen patients on the drug (see Table 2) has shown the following:

1. All patients tested had a positive ANA.
2. Two patients developed an immediate reaction to the drug – drug fever.
3. Fourteen out of sixteen had no suggestion of a lupus diathesis.
4. Two patients developed the lupus syndrome –
   (a) Case #2 arthralgias and positive ANA to 1/1024.
   (b) Case #16 (reported)
5. The drug was stopped in two additional patients because of a rising ANA titre.

Previously my obtaining an ANA was haphazard, usually within two months of initiating therapy and then again only if symptoms appeared. There are few suggestions in the literature concerning the predictive value of the ANA as a guide to the use of procainamide. The only two I could find[3,4] suggested a baseline determination and repeated testing every three to four months or when symptoms appeared.

My own method now is to obtain a baseline determination as it has been shown that 16% of healthy people over the age of sixty (mostly females) have positive

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The Early Recognition of Scoliosis

PAUL M. BEEGEL, M.D., F.A.C.S.* and VICTOR M. PARISIEN, M.D., F.R.C.S. (C)**

Scoliosis is defined as one or more lateral-rotatory curves of the spine. Failure to recognize this condition in its early stages may lead to cosmetic distortion, severe interference with pulmonary and cardiac function, early disabling chronic backache, and on occasion even paraplegia. Our hope is to present a paper which will allow those not overly familiar with this condition in its very early stages and with minimal signs to be more readily recognizable.

To the average physician, this seems a rare condition. To the orthopaedic surgeon it is too common, and recognized much too late. Few studies of the incidence of this disease have been made and these are not conclusive since they do not include adequate numbers of diversified population. However, these studies do indicate that the incidence in school children may be as high as 10+% in some areas. If this figure is even near correct and projected to the Maine population, we should be able to find 30,000 cases in this State. Females have the condition ten times as frequently as males in the idiopathic form which is the most common one and to which our attention is primarily directed.

From the standpoint of types, there are four in general classification: (1) Idiopathic, (2) Congenital, (3) Paralytic, and (4) Acquired. The etiology of the three latter types is self-explanatory. They usually give rise to severe obvious curves which usually are recognized and treated early. Idiopathic scoliosis is slower, usually always asymptomatic and insidious in onset.

Recently, the Scoliosis Research Society has recommended that this particular type of scoliosis be called Genetic Scoliosis. This follows investigations which indicate that between 50% and 80% of all these children have direct blood line relatives with this condition or its analogous condition kyphosis. The condition can be found in parents, grandparents, uncles, aunts, and most important – siblings. From the above, it follows that the first point of contact may well be the observance of the condition in *ANY* family member. If an adult, questions followed by examination of children in all branches of the family should be instituted.

The condition in its idiopathic or genetic form is divided into three age groups: (1) Infantile (birth to age 3), (2) Juvenile (age 3 to 10 years), and (3) Adolescent (age 10+ years). The infantile type is rare in this country, is much more benign, and most, BUT NOT ALL require only simple observation to resolution. The juvenile groups should form the largest group of new patients. We

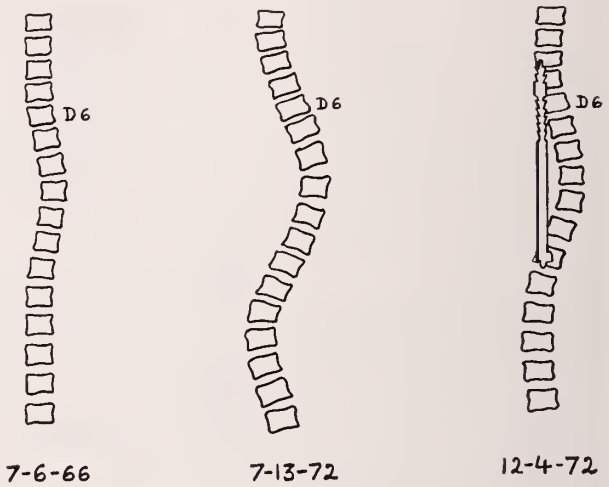

Fig. 1. Line drawings of the x-rays on DK, female, now age 15 years. The x-ray of 7/6/66 is one found on this patient age 9 in her microfile when she was admitted to Crippled Children's Clinic in July of this year. The minimal curve noted on the x-ray had not been followed up and on admission measured 59°. With surgical correction using the Harrington rod technique and fusion, this curve was corrected to 22°. While this sounds remarkable, the fact of the matter is that had this curve been followed adequately it need never have been allowed to come to this deforming degree and might very well have never needed surgery. Incidentally, this patient has three siblings, all of whom have scoliosis who hopefully will be able to be treated throughout by conservative means.

believe that this condition does not usually start in adolescence but most probably starts in the early juvenile formative years and progresses quickly during the rapid and extensive growth spurts of adolescence. Thus, it is our feeling that the adolescent classification in most instances is nothing more than a classification based on the age of discovery rather than the time when the curvature actually begins. It should be remembered however, that progression in both juvenile years and adolescence may proceed rapidly and to severe rigid structural deformities causing devastating cosmetic appearance, and severe interference with respiratory and cardiac function. Such rigid curves are difficult to treat, require mostly surgical intervention, may not be correctable to completely acceptable cosmetic appearance, increase the difficulties and complications of surgery, lay the patient up for prolonged periods of time, increase costs of treatment, and in final analysis are in most instances unnecessary.

The two most common sources of case findings are:

1. The child with poor posture.

Many teenagers and younger groups tend to cultivate a "round shouldered attitude" as part of their teen-culture. Parents may bring such a child in asking if there is not some type of brace which will force the child out of this

Continued on Page 57

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**Assistant Orthopaedic Surgeon, Central Maine General Hospital, Lewiston, Maine 04240.

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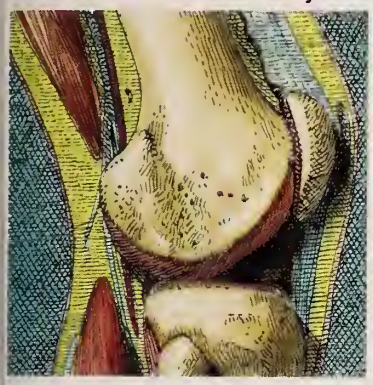

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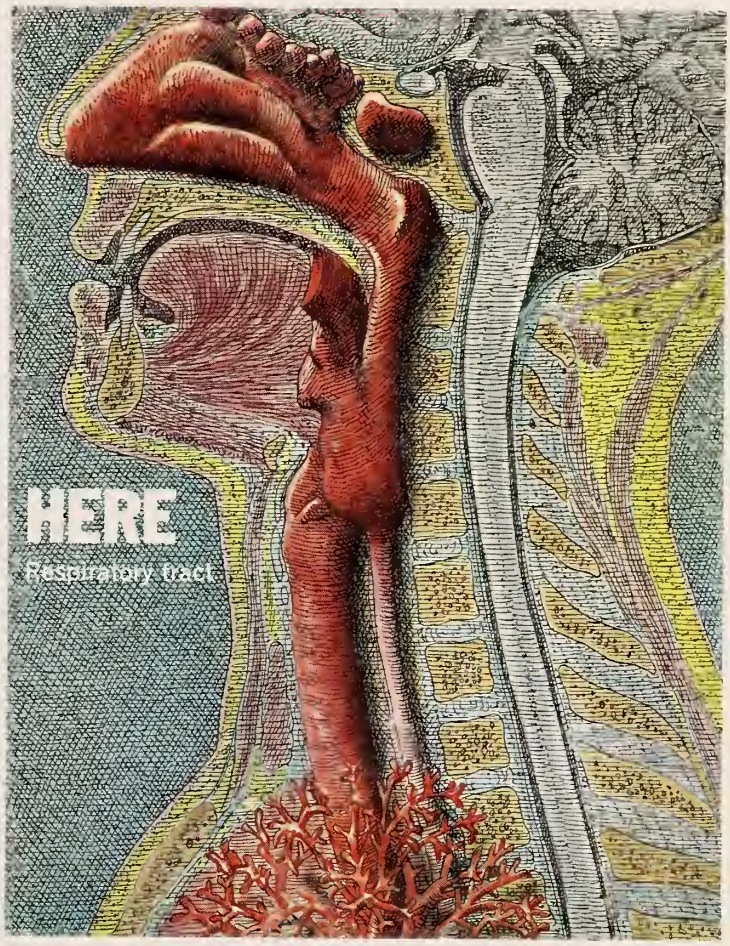

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Indications: Lomotil is effective as adjunctive therapy in the management of diarrhea.

Contraindications: In children less than 2 years, due to the decreased safety margin in younger age groups, and in patients who are jaundiced or hypersensitive to diphenoxylate HCl or atropine.

Warnings: Use with caution in young children, because of variable response, and with extreme caution in patients with cirrhosis and other advanced hepatic disease or abnormal liver function tests, because of possible hepatic coma. Diphenoxylate HCl may potentiate the action of barbiturates, tranquilizers and alcohol. In theory, the concurrent use with monoamine oxidase inhibitors could precipitate hypertensive crisis.

Usage in pregnancy: Weigh the potential benefits against possible risks before using during pregnancy, lactation or in women of childbearing age. Diphenoxylate HCl and atropine are secreted in the breast milk of nursing mothers.

Precautions: Addiction (dependency) to diphenoxylate HCl is theoretically possible at high dosage. Do not exceed recommended dosages. Administer with caution to patients receiving addicting drugs or known to be addiction prone or having a history of drug abuse. The subtherapeutic amount of atropine is added to discourage deliberate overdosage; strictly observe contraindications, warnings and precautions for atropine; use with caution in children since signs of atropinism may occur even with the recommended dosage.

Adverse reactions: Atropine effects include dryness of skin and mucous membranes, flushing and urinary retention. Other side effects with Lomotil include nausea, sedation, vomiting, swelling of the gums, abdominal discomfort, respiratory depression, numbness of the extremities, headache, dizziness, depression, malaise, drowsiness, coma, lethargy,

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Each tablet and each 5 ml. of liquid contain:
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(Warning: may be habit forming)
Atropine sulfate ..... 0.025 mg.

anorexia, restlessness, euphoria, pruritus, angioneurotic edema, giant urticaria and paralytic ileus.

Dosage and administration: ***Lomotil is contraindicated in children less than 2 years old.*** Use only Lomotil liquid for children 2 to 12 years old. For ages 2 to 5 years, 4 ml. (2 mg.) t.i.d.; 5 to 8 years, 4 ml. (2 mg.) q.i.d.; 8 to 12 years, 4 ml. (2 mg.) 5 times daily; adults, two tablets (5 mg.) t.i.d. to two tablets (5 mg.) q.i.d. or two regular teaspoonfuls (10 ml., 5 mg.) q.i.d. Maintenance dosage may be as low as one fourth of the initial dosage. Make downward dosage adjustment as soon as initial symptoms are controlled.

Overdosage: Keep the medication out of the reach of children since accidental overdosage may cause severe, even fatal, respiratory depression. Signs of overdosage include flushing, lethargy or coma, hypotonic reflexes, nystagmus, pinpoint pupils, tachycardia and respiratory depression which may occur 12 to 30 hours after overdose. Evacuate stomach by lavage, establish a patent airway and, when necessary, assist respiration mechanically. Use a narcotic antagonist in severe respiratory depression. Observation should extend over at least 48 hours.

Dosage forms: Tablets, 2.5 mg. of diphenoxylate HCl with 0.025 mg. of atropine sulfate. *Liquid,* 2.5 mg. of diphenoxylate HCl and 0.025 mg. of atropine sulfate per 5 ml. A plastic dropper calibrated in increments of ½ ml. (total capacity, 2 ml.) accompanies each 2-oz. bottle of Lomotil liquid.

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Adverse Reactions: Drowsiness, excessive dryness of nose, throat or mouth; nervousness; or insomnia. Also, nausea, vomiting, epigastric distress, diarrhea, rash, dizziness, weakness, chest tightness, angina pain, abdominal pain, irritability, palpitation, headache, incoordination, tremor, dysuria, difficulty in urination, thrombocytopenia, leukopenia, convulsions, hypertension, hypotension, anorexia, constipation, visual disturbances, iodine toxicity (acne, parotitis).

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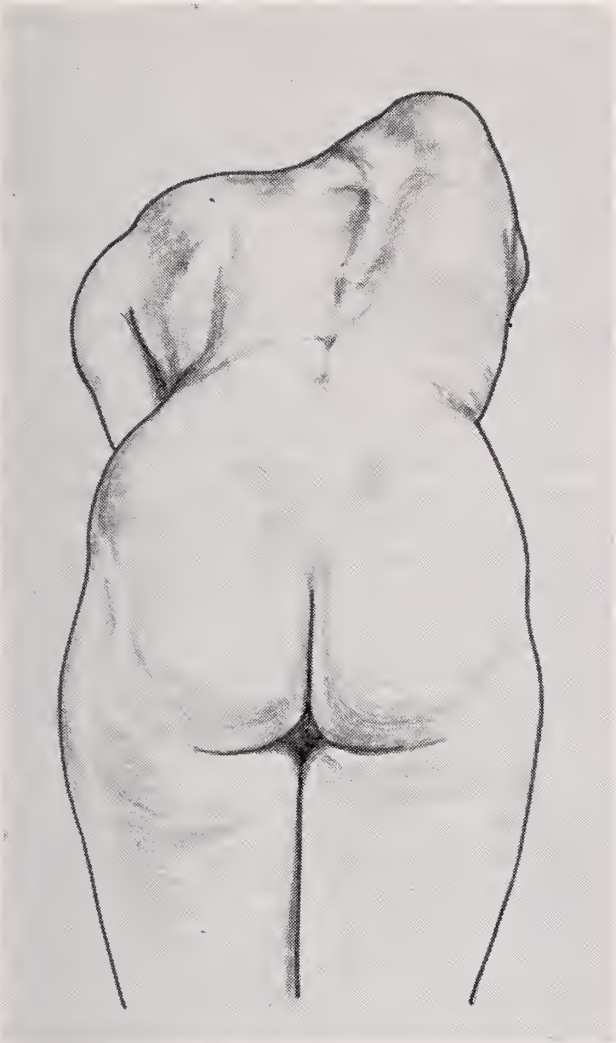

Fig. 2. The figure shows the body bending over as described in the text. Note the "razorback" rib hump and the attempt to illustrate a lumbar hump, both of which are markedly exaggerated for illustrative purposes. If humps in a patient are as illustrated, the problem is severe and in most instances beyond conservative lines of treatment. In seeking early diagnosis, the deformities must be found at a much lesser degree of clinical visibility than illustrated.

Fig. 3. Note the neck line in this illustration and its asymmetry at the left cervicoscapular junction. Note the asymmetry of the hang of the arms in relation to the body, and note also the attempt to illustrate the prominence of the scapula on the right side in relation to that on the left. Note also the folding in of the right flank in relation to the left.

posture. Such children should be examined with clothes off to see the spine. Motion of the back must be checked and x-rays may be necessary to rule out Scheurermann's Disease or juvenile kyphosis (a treatable condition). They should also be checked out of clothing to make sure that the "round shouldered" posture is not due to a "razorback" rib hump (Fig. 2) with a moderate to severe scoliotic curve. Many of these children may be referred by school doctors, nurses, teachers, and physical education instructors who have the opportunity to observe a child from a more objective point of view than most parents are usually capable of doing. Remember, the disease is usually asymptomatic; it is insidious in onset and progression; parents do not usually see the children out of their clothes from the time they begin taking their own baths and dressing themselves; and lastly, the age of "false modesty" occurs in the child at that age when the disease is most apt to progress rapidly.

2. The child whose mother complains that she cannot get clothes to fit properly.

This is usually the female of the group. Mother will complain that making clothes for the child is difficult because she "does not fit the pattern" or that clothing purchased must be altered to make seams and hems hang properly and evenly. This child may have a scoliosis which causes this difficulty or may have a pelvic obliquity (tilt of the pelvis) creating the asymmetry, and which will in turn create a spinal curvature.

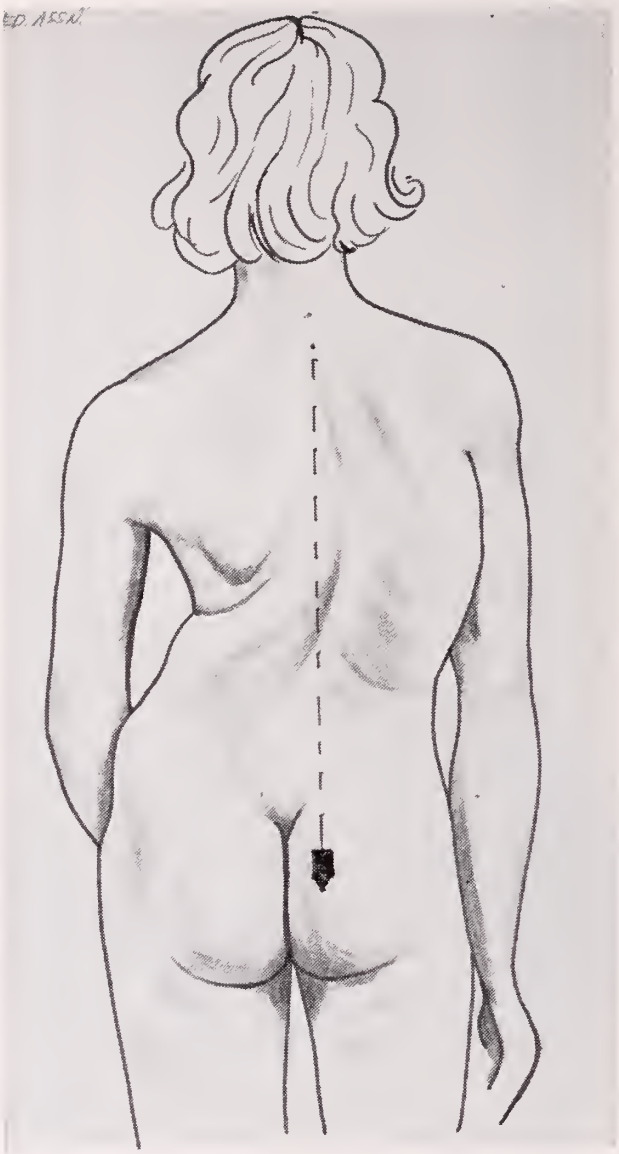

Fig. 4. In this we have attempted to illustrate a number of factors. Note the following: A list to the right; assuming right handedness, the elevation of the right shoulder; elevation of the left pelvic brim; the plumb line off to the right with the plumb to the right of the natal fold; the prominence of the left hip; and the distortion and prominence of the right scapula.

Examining a Child

For proper examination, the child should be unclothed. The patient should disrobe to bra and panties or to shorts. For posterior examination, the bra should be open to expose the back and panties or shorts pulled down far enough to see the natal fold. For anterior exam, the bra is removed and panties or shorts lowered sufficiently to see and palpate the anterior superior iliac spines. The child should stand erect with weight evenly placed on both feet with the feet a few inches apart for balance, the head erect and looking straight ahead, and the body relaxed in comfortable stance, arm hanging at the sides. Invite parents to be present for this examination and point out and explain the method of exam and findings to them as you go along. This will teach them what to look for and also teaches the child as well. You may find it amazing but many patients are referred by teenage patients already under care.

Now look at the neck line. A prominence of the neck on one side at its angle with the shoulder or fullness on one side may indicate a high thoracic curve (Fig. 3). These curves do not have to be severe to produce this deformity.

Does the patient appear to list to one side? If necessary, hang a plumb line from D-1 spinous process (Fig. 4). If it does not match the natal fold is it because of list, curvature, or pelvic obliquity tilting the body?

Do the arms hang symmetrically at the sides (Figs. 3 and 4)? Is there a difference in the amount of space between the arm and the body on one side? If there is, does there appear to be more indentation of the flank on one side than the opposite side (Fig. 4)?

Does the pelvis appear to be tilted (Fig. 4)? Does one hip appear to be more prominent on one side as opposed to the other when weight is evenly distributed (Fig. 4)? Check the level of the tops of the iliac crests and the anterior superior iliac spines. If these are not even and do not form an imaginary line parallel to the floor, then leg length should be checked for the possibility of a short leg.

Are the breasts symmetrical in the female? If not, look for asymmetry of the anterior chest wall. Asymmetry of the breasts in the teenage female may be normal. Asymmetry of the rib cage is not, and indicates probable scoliosis.

Is the shoulder of the dominant side elevated (Fig. 4)? Normally it is level with or slightly lower than the non-dominant side.

Run the fingers down over the spinous processes for the full length of the spine. Make sure to check the lumbar spine particularly around the lumbosacral junction carefully. The primary lumbar curve if minimal may not be visible and is the most difficult of all to recognize even by those well trained. If in any doubt – x-ray.

Is there any prominence of one scapula over another (Figs. 3 and 4)? If this is not obvious from routine clinical examination, have the child bend forward allowing the arms and head to hang down and look tangentially across the top of the back. Any minimal rib hump or scapula will then become much more obvious (Fig. 2).

This same commentary applies to prominence of one or the other side of the lumbar spine with prominence of the lumbar musculature. The examination is carried out in the same way except that the patient bends further forward. Note that patients with double or triple curves may well have both dorsal and lumbar humps present at the same time but on opposite sides. It is rotation in the curve which causes this distortion.

Obviously when looking for these deformities, we are not talking about the patient who has a 50 or 60 degree curve, a 4-inch rib hump, a 2-inch lumbar hump, and stands 2 inches off center on plumb line. His problem is obvious. We are looking for the patient with MINIMAL

curvature. Therefore, the signs and findings outlined above will be minimal and the examination must be conducted by the most careful observation. In practically all instances, if the practitioner will train himself to look for these telltale signs, he will find that in a short time these signs become obvious in many instances even in the fully dressed patient, to the point that he will request complete disrobing for more adequate exam.

Summary and Conclusions

1. Primary early diagnosis is made by those who think of and look for this disease which is far more common than realized.

2. A method of examination has been outlined.

3. Examination is primarily a matter of trained observation.

4. Where there is ANY question, x-rays should be taken. The minimum x-ray examination should consist of an AP standing and an AP supine of the entire dorsal and lumbar spine. Where the patient is round shouldered, a lateral dorsal spine film should be taken to rule out juvenile kyphosis. Any other unusual spinal abnormality or suspected abnormality should be x-rayed to rule out other conditions which may lead to severe scoliotic changes.

5. In the presence of other congenital anomalies, clinical examination of the back should be carried out at regular intervals – at least twice a year until certain that growth has been completed and no curvature has evolved.

6. Do your examination and indicate to both parents and patient how you are conducting the exam and what you are looking for. Point out the findings so that those who are involved can see and understand.

7. Request parents to discuss with their siblings the fact that this disease exists in the family and that all children should be examined for this condition. The fact that "idiopathic" scoliosis is now termed genetic scoliosis has basis in fact and all members of the family with children should be alerted to this.

8. Do not presume that in late teen years growth may have stopped. If growth is not complete, such a presumption may be disastrous. More recent concepts have extended the age during which growth may occur and if scoliosis is present and growth is not complete, curves may get worse. Even experts do not agree completely on this subject and frequently have trouble making decisions on individual patients. If in doubt, continue to see and observe the patient.

9. Do learn to measure curves by the Cobb Method of measurement. It is difficult even for the most experienced to look at an x-ray and estimate the degree of curvature. Where a curve progresses to clinical visual acceptance of increase, treatment is obviously indicated. However, the patient who only increases the small increments (2-5°) also needs treatment and his steadily insidious increase may not be obvious unless measurements are properly taken, recorded, and compared.

10. Do follow up on any patient whose x-rays show ANY curvature for ANY reason. Failure to do so may initiate a disastrous sequence of events for the future of the patient. Repeat films at reasonable intervals (3-6 months) properly measured and compared with previous films will assist in proper diagnosis at an early stage and allow for an easier line of therapy where necessary.

If you do not wish to, or do not feel qualified to, take on the full job of following these patients adequately, refer them to one of the several centers in Maine where trained personnel, equipment, and therapeutic management procedures are available. Frequently these patients will need a team approach with the assistance of trained physiotherapists, brace makers, nurses, social workers, as well as doctors, and members of the family. If minimal lesions are to be kept minimal or corrected, they must be recognized early, treated vigorously, and have adequate followup. Only in this way can we prevent the crippling deformity, destruction of middle years health and activities, and ugly cosmetic problems.

Acknowledgement

We wish to thank C. F. Miller, M.D., Chief Radiologist, Central Maine General Hospital, for drawing the diagram and illustrations.

References

Keim, Hugo A.: "Scoliosis," Clinical Symposia; 24: 1-32, 1972.

Blount, Walter P.: "Early Recognition and Prompt Evaluation of Spinal Deformity," The Wisconsin Medical Journal; 68: 245-249, 1969.

Brooks, Leon; Gerber, Erwin; Mazur, Harold; Brooks, Rose; Nickel, Vernon L.: "The Epidemiology of Scoliosis – A Prospective Study," Orthodaedic Review; 1: 17-23, 1972.

Continuing Professional Education

RICHARD T. CHAMBERLIN, M.D.* and FLOYD B. GOFFIN, M.D., F.A.C.S.**

"From what point of view is the problem of medical education to be studied and presented?"[1] Thus, Flexner began a new era in medical education four or five decades ago. Many of the changes he proposed have been instituted, so that today we no longer find "diploma mills" among the listings of medical schools in the United States. On the other hand, "it is the most expensive . . . of medical education, because of its emphasis on the teaching laboratory in the basic sciences and small group teaching at the bedside in the clinical years."[2]

The question originally asked[1] about medical education may be placed to the issue of continuing medical education. In recent years this aspect of "training" has come under study, been experimented upon, prescribed or insisted upon. To some it became an exercise in study for its own sake; to others an interesting laboratory challenge; to those for whom it was prescribed it tasted sweet or felt like a harsh cathartic. With the increasing attention of consumer, government and third party payors it is becoming increasingly apparent that continuing education is here to stay. It behooves the medical profession to take charge of its further development and management so that its intended aim – to improve patient care – shall be realized!

This is not to say that patient care is not now good. It merely asserts continuing medical education should be equated with continuing patient care improvement.

The first step in achieving any concepts about postgraduate medical education must lie in a definition of the problem. To this purpose the A.M.A. sponsored the Third Conference of State Medical Association Representatives on Continuing Medical Education in Chicago, October 24-26, 1972. The authors of this paper represented Maine. It is the purpose of this paper to report the sum and substance of that meeting to the readers of the Journal.

Six states (Oregon, Arizona, Pennsylvania, New Jersey, Massachusetts, and Florida) now require or are in the process of implementing a requirement of continuing medical education as a basis of continued society membership. Arizona requires education credits as a prerequisite to state licensure.

The American Academy of Family Physicians has a requirement of continuing medical education for membership.

The A.M.A.'s Physician Recognition Award permits documentation of continuing medical education in all areas of medicine.

These trends are under close scrutiny by most states, specialty organizations and groups interested in the quality of patient care.

The Chairman of the Oregon Medical Association[3] pointed out that the State of Oregon has approximately two thousand members. Oregon has spelled out in detail[4] its requirements for membership. As the last month of the first cycle of their membership requirement began, all but fifty-three members of the society had reported their continuing medical education activity satisfactorily. In the final month, fifty-three hearings were held on those who had chosen not to report their continuing medical education activities. In some of these fifty-three, the hearings resulted in an extension of the time allowed for the reporting. However, in the final analysis, there were eleven members who were finally dismissed from membership. Since that time, three have been reinstated. During the time that all this occurred and eight members were lost to the society by this mechanism, there was an overall eight percent gain in membership of the Oregon Medical Association.

In summation, in those states where the requirement has gone into effect, although it has caused some problems, there has not been a feeling that the requirement for continuing medical education has been a detriment to the state society.

The President-elect of the American Society of Internal Medicine feels that "peer review is evolving from a concern about cost to a concern about quality of medical care. Through a comparison of actual performance of patient care with previously determined standards for optimal care, peer review will uncover the material for continuing medical education to use in helping physicians to improve their performance."[5]

The subject of accreditation for continuing medical education by state medical associations was described by a spokesman for the California Medical Association.[6] The guidelines that California has developed are very similar to those used by the A.M.A. in its process of accrediting education programs and it has been felt by the California group that the process of accrediting programs locally has done much to upgrade the quality of programs within the state as well as to allow for the more easy access to accredited programs by the membership of the California Medical Association. The details of the accrediting process are too great to go into this report but the committee on continuing medical education of the Maine Medical Association will be reviewing these in an attempt to establish an accrediting procedure in the State of Maine.

SUMMARY AND RECOMMENDATIONS

1. The Maine Medical Association should reaffirm its belief in the basic educational principle that each in-

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dividual physician has an obligation to himself and his profession, as well as a responsibility to his patients to renew and update his medical knowledge in a process of continuing education. It is recognized that, for effective continuing medical education, the means may be varied and requirements uncertain, but the objective desirable. Therefore, the committee on continuing medical education will be working towards a requirement, that in order to maintain one's membership in the Maine Medical Association, each physician will be required to report his educational activities, but not necessarily set a limit as to the number of hours or the types of these activities at this particular point in time. It is hoped that this type of program will develop into an effective system whose study and modification will lead to appropriate future requirements.

2. Although the procedure may be somewhat cumbersome, it is recommended that the Maine Medical Association continue to pursue the establishment of an accrediting procedure. Details of this will have to be worked out.
3. That the Committee on Continuing Medical Education assist the Peer Review Committee in shaping the emphasis of peer review onto the quality of medical care, rather than merely claims review and cost containment.

REFERENCES

1. Flexner, A.: Medical Education in the United States and Canada. The Carnegie Foundation for the Advancement of Teaching, Bulletin No. 4, New York, 1910.
2. Medical Schools for the Modern World. John Z. Bowers, ed., The Johns Hopkin Press, Baltimore and London, 1970.
3. Pennington, M.: Conference Communication.*
4. Policy Statement relating to the Oregon Medical Association Postgraduate Education Program, 1971, as adopted by the House of Delegates.
5. Felch, W.: Conference Communication.*
6. Opfell, R. W.: Conference Communication.*

*The Third Conference of State Medical Association Representatives on Continuing Medical Education, Chicago, October 24-26, 1972.

ACCELERATED HYPERTENSION SECONDARY TO ORAL CONTRACEPTIVES

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9. Harris, P. W. R.: "Malignant Hypertension Associated with Oral Contraceptives." Lancet; 466-467, 1969.
10. Woods, James W.: "Oral Contraceptives and Hypertension." Lancet; 653-654, 1967.
11. Goodlin, Robert C., Waechter, Vera: "Oral Contraceptives and Blood Pressure." Lancet, (Letters to the Editor); 1262-1263, 1969.
12. Laragh, John H., Sealey, Jean E., Ledingham, John G. G., Newton, Michael A.: "Oral Contraceptives (Renin, Aldosterone, and High Blood Pressure)." Journal of the American Medical Association; 201, 98-102, 1967.
13. Kunin, C. M., McCormick, Regina, Abernathy, James R.: "Oral Contraceptives and Blood Pressure." Archives of Internal Medicine; 123, 363-365, 1969.
14. Saruta, Takao, Saade, Guillermo A., Kaplan, Norman M.: "A Possible Mechanism for Hypertension Induced by Oral Contraceptives." Archives of Internal Medicine; 126, 621-626, 1970.
15. Tyson: "Oral Contraception and Elevated Blood Pressures." American Journal of Obstetrics and Gynecology. 100; 875, 1968
16. Ndeti, C. S., Horrobin, D. F., Burstyn, P. G., Hopcraft, M. M.: "Salt to Lower Blood Pressure." New England Journal of Medicine. (Correspondence); 286; 782-783, 1972.

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Medicaid Adds Child Screening Emphasis

John E. Fickett*

The Maine Medical Assistance Program (under Title XIX of the Social Security Act), popularly known as "Medicaid," is taking on a new emphasis. The Division of Medical Assistance, which is the Medicaid administrative unit, is aggressively promoting child screening services, in addition to the other current services of the Medical Assistance Program.

Currently, the target group is all of the children under age 7 eligible for Medicaid in Maine, numbering about 17,000. Starting July 1, 1973 all eligible children under age 21 will be included in the screening emphasis. The total then will number approximately 50,000 (about 13% of total Maine population under age 21).

The official title of the new promotion is "Early and Periodic Screening, and Diagnosis and Treatment" (EPSDT) which was promulgated in a Federal regulation (45 CFR 249.10) effective February 7, 1972. The regulation required Medicaid states to include dental care, eyeglasses, and hearing aids as part of their services for children. Maine began to provide those services for eligible children under age 7 on that date, and on July 1, 1973 will extend the three additional services to all eligible children under age 21.

Our Division is making agreements with various agencies so that EPSDT services will be available statewide. We are working cooperatively with the State Comprehensive Health Planning agencies in qualifying such screening agencies.

However, it is recognized that many medicaid children have a family physician. Of course, our Division does not want to do anything to disturb the doctor-patient relationship. Therefore, those children who are identified by screening agencies as being under medical care, or desiring screening or a physical examination by their family physician, will be reported to our Division as being accounted for. In such cases, our Division will contact the family physician and request his cooperation in helping us to be informed as to what the child's health status may be.

As part of the EPSDT services, we are establishing a health record for every child. Those children who are found, by screening, to have apparent physical and/or mental defects will be referred by the screening agencies to the physician of their choice for definitive diagnosis and treatment as indicated.

The EPSDT emphasis will include outreach to connect every eligible child with his physician (or where none, with a screening agency). Children screened will be followed up after referral to a physician, to ascertain whether the child kept appointments, and to encourage such without intrusion into the child's private arrangements. A child or his parent (or adult caretaker) has the right to refuse participation in EPSDT services but screening agencies will endeavor by personal contact to inform parents in a manner that will engage participation by as many children as possible. Medicaid will pay for transportation of children under age 7 (under 21 effective 7/1/73) to EPSDT services when not otherwise provided.

As an average, approximately 50% of eligible children actually receive at least one Medicaid service in a given year. Many of the remaining 50% (or about 25,000 children under age 21) may have an undiagnosed and untreated physical or mental defect. Screening or medical review by the family physician could turn up these problems. These children are eligible to obtain such services without charge to them or their parents.

The objective of EPSDT is to provide a high quality care of adequate scope in a timely manner so as to stimulate the use of preventive health services with the aim of enhancing the health of children in the State of Maine. We join the participation of the physicians of Maine in this lofty goal.

Further information about EPSDT may be obtained from the Division of Medical Assistance, Department of Health and Welfare, State House, Augusta, Maine 04330, or telephone 289-3846 (area code 207).

*Director, Division of Medical Assistance, Maine Department of Health and Welfare, Augusta, Maine 04330.

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Maine Heart Association Notes
20 Winter Street, Augusta, Maine

Sustained Isometric Handgrip — A Useful Bedside Maneuver

Nallan C. Ramakrishna, M.D.*

The advent of each new cardiovascular diagnostic technique has led to a broadened foundation for verification and refinement of the technique of physical examination enabling the astute physician to utilize physiological principles to enhance his diagnostic acumen. Thus, for example, cardiac catheterization has helped to define the relationships between variations of splitting of the second heart sound and intracardiac pressures. Recognition and characterization of wall motion disorders with the radarkymogram, with isotope angiocardiograms, and with left ventricular cineangiograms has facilitated clinical detection of dyskinesis in patients with acute myocardial infarction and hence provided the clinician with an additional important diagnostic sign of this disorder.

In part as an outgrowth of exercise testing and pharmacologic stress testing of the cardiovascular system, a new technique, isometric handgrip, has been utilized to further enhance the value of physical examination of the heart. Isometric handgrip, a simple bedside physiological stress test, can be utilized to characterize and differentiate the origin of several murmurs and to detect impaired ventricular performance.

Sustained isometric handgrip promptly elevates systemic arterial pressure, increases heart rate, and augments cardiac output. These changes regress quickly when handgrip is released. The physiological response to sustained isometric handgrip appears to be mediated by reflex mechanisms. Resulting changes in the peripheral vascular resistance impose an acute stress on the left ventricle by increasing afterload.

The test is performed with the use of a commercially available Handgrip Dynamometer. The patient is asked to squeeze the Dynamometer with the maximum force he can exert. After the extent of maximum voluntary contraction has been determined and the patient has rested, he is then instructed to initiate and maintain handgrip at 25 to 50% of the maximal force for a period of 2 to 3 minutes. Graphic recordings of heart sounds, the electrocardiogram, or hemodynamic measurements can be obtained to supplement auscultation performed during the interval in which handgrip is sustained. When a Dynamometer is not available, the test can be performed by asking the patient to squeeze the examiner's hand for the desired interval.

Although the test is safe, caution should be exercised when it is utilized in patients with coronary artery disease. The acute stress imposed can lead to the precipitation of angina or serious arrhythmias. Hence, testing such patients should be performed with concomitant monitoring of the electrocardiogram and emergency equipment should be available for correction of serious arrhythmias.

Isometric handgrip is helpful in differentiating systolic murmurs of aortic and mitral origin. The murmur of aortic stenosis becomes less intense because the gradient across the aortic valve is reduced. In contrast, the holosystolic murmur of mitral regurgitation increases in intensity due to the increase in regurgitation secondary to increased afterload.

In patients with the prolapsing posterior mitral leaflet syndrome (Click-murmur syndrome), the late systolic murmur may be illicited by isometric handgrip. If the murmur is already present at rest, it may intensify and its duration prolonged by this maneuver. In patients with this syndrome, the click often moves nearer to the first heart sound during isometric handgrip.

Handgrip increases the intensity of the diastolic murmur of aortic regurgitation. Hence, it is useful in distinguishing aortic regurgitant murmurs from pulmonic regurgitant murmurs which are not altered by handgrip.

Handgrip may intensify the diastolic rumble of mitral stenosis because tachycardia reduces diastolic filling time and therefore the pressure gradient across the mitral valve increases.

Sustained isometric handgrip is helpful in distinguishing the Austin Flint murmur associated with aortic regurgitation from the presystolic rumble of mitral stenosis. The Austin Flint murmur in patients with moderately severe aortic regurgitation is presystolic in timing. As regurgitation becomes severe, the murmur becomes middiastolic due to the increase of left ventricular end diastolic pressure resulting in closure of the mitral valve before the end of the diastolic period. Thus, with the hand grip test, the presystolic murmur of the Austin Flint type shifts to middiastole in contrast to the murmur of mitral stenosis which becomes louder without change in timing.

In patients with either ischemic heart disease or

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Prepared by the Maine Heart Association for this Journal.

—News From Blue Cross and Blue Shield—

COMPANION PLAN BENEFITS INCREASED

Maine Blue Cross and Blue Shield has expanded its Companion Plan I (Medicare supplement) coverage to absorb the higher Medicare deductibles and co-insurance payments for hospital and medical care that took effect January 1, 1973.

As announced by the U.S. Department of Health, Education and Welfare, the changes in Medicare Part A effective January 1 make elderly persons responsible for the first $72 of their hospital bills instead of the present $68. In addition, the co-insurance charge which they must pay for the 61st through the 90th day of hospitalization has been increased to $18 a day from the previous $15, and Medicare co-insurance charges applicable to the 60 day "lifetime reserve" has been increased to $36 a day from $34.

The supplementary medical insurance deductible in Medicare Part B was increased from $50 to $60 with the co-insurance remaining at 20%. These changes are covered according to the benefits of the Companion Plan.

Richard F. Nellson, President of Maine Blue Cross and Blue Shield, explained that approximately 66,000 Companion Plan subscribers have these higher Medicare deductibles and co-insurance costs covered automatically at no extra membership charge.

For additional information about the Companion Plan, write to the Portland Office, 110 Free Street, Portland, Maine 04101.

COORDINATED HOME HEALTH CARE COVERS WIDE AREA

Coordinated Home Health Care, which provides professional health services and supplies for eligible subscribers when they require a combination of hospital-type services in their homes, covers a wide area in Southern Maine. Efforts are being made by officials of Maine Blue Cross and Blue Shield to expand this coverage to other areas of the State; but right now, the cities and towns included in the program are Bridgton, Brownfield, Cape Elizabeth, Chebeague Island, Cumberland, Denmark, Falmouth, Freeport, Fryeburg, Gorham, Harrison, Lovell, Naples, North Yarmouth, Peaks Island, Portland, Pownal, Scarborough, Sebago, South Portland, Stow, Sweden, Westbrook and Yarmouth.

SUSTAINED ISOMETRIC HANDGRIP — A USEFUL BEDSIDE MANEUVER

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primary myocardial disease atrial or ventricular gallops which may be absent at rest can be illicited by isometric handgrip. The acute stress induced by the maneuver results in the production of filling sounds, i.e., the atrial and/or ventricular gallops, because of decreased ventricular compliance.

In summary, isometric handgrip is a simple diagnostic maneuver which can provide useful information at the bedside and in the diagnostic laboratory. It is of value in detecting valvular and myocardial disease and in elucidating its nature and extent. It represents an excellent example of the application of physiological principles in the clinical setting to improve cardiovascular diagnosis.

County Society Notes

WASHINGTON

The regular meeting of the Washington County Medical Society was held on October 12, 1972 with eleven members present, including guests.

Minutes of the last meeting were read and approved.

There will be a combined meeting of Hancock-Washington-Waldo County Societies on November 1, 1972 at Jasper's Restaurant in Ellsworth, Maine. Dr. George W. Wood, III, President of the Maine Medical Association will be one of the speakers during a business analysis. Following lunch, there will be a panel series.

Dr. Robert G. MacBride brought up the name of Dr. Lawrence Moore as a member of the Society. It was moved, seconded and passed that Dr. Moore be elected a member of the Society.

Dr. G. Bernard Shaw, President of the Washington County Medical Society, then introduced Kenneth Cole of Bangor, Maine of the PENQUIST Community Action Agency. With him was Charlotte Adams, R.N. Their intention is to come into Washington County to examine children under Medicaid from 0 through 6 years of age. They expect to cover such things as visual, hearing, immunizations, B.P. and urine. The local physicians are to give the complete physical examinations. Nurses will do the Hgb. and urinalysis. If a child has had such an examination within one year's time, a new physical will not be done. It is hoped that at each session, they will be able to handle about 20 children during a 4-hour period.

Charlotte Adams, R.N. spoke relative to results of some of their findings and said that in some of the poorer areas, many of the children had never had such an examination. Mrs. Adams said that usually it would cover nine items in the physical examination. Usually, each clinic would be staffed with a physician, one nurse and two aides.

There was considerable discussion by members of the Washington County Medical Society in regard to the above program. It is the hope of the Society that Mr. Conridge of the Down East Health Service would be admitted to the project, as an administrator, so that we might have better control of what is going on. It was voted that the members of the Board of Directors of the Down East Health Service would coordinate the plans with the PENQUIST Community Action Agency in this particular program and would also look into Health Start.

Meeting adjourned at 9:30 p.m.

KARL V. LARSON, M.D., *Secretary*

KENNEBEC

The Kennebec County Medical Association met on Thursday, September 21, 1972 at the Jefferson in Waterville, Maine. Following a social hour and dinner, (served for the 47 members and guests) a business meeting was opened by the President, Dr. Francis J. O'Connor. The minutes of the last meeting were read and accepted. Dr. Bhupendra P. Mepani of Augusta was elected to full membership. A communication was read by Mr. Sumner H. Lipman, President of Kennebec County Bar Association, proposing a joint medical malpractice plan to be sponsored by our two associations. This communication was referred to the Council which will act as a steering committee on this matter. Further discussion of this proposal is scheduled for our October meeting and for the joint meeting of our two societies scheduled for November 1972.

A letter from Dr. Richard T. Chamberlin was read, which asked for discussion and recommendations from our Association in the matter of participation in the Annual Diabetes Detection Drive, sponsored by the Diabetes Committee of the Maine Medical Association. There was considerable discussion of this matter, including comments by physicians who had served as Kennebec County Diabetes Drive Chairmen. As a result of this discussion, a motion was proposed by Dr. Sewall, directing the Secretary to write a letter to the Diabetes Committee of the Maine Medical Association indicating that the Kennebec County Medical Association feels that the amount of effort and money spent in the Annual Diabetes Drive to date is not justified by the paucity of useful results accrued to date.

Dr. O'Connor then introduced Dr. Richard C. Britton, Chief Surgical Service, Maine Medical Center, who spoke on the subject of "Selective Portal Decompression." Dr. Britton discussed the neurological sequelae of conventional shunt procedures and gave a most thorough and beautifully illustrated review of the advantages of the selective, central type of spleno-renal decompression for portal hypertension. The meeting was adjourned at 9:20 p.m. by Dr. O'Connor.

The Kennebec County Medical Association met at the Holiday Inn, Augusta, Maine on Thursday, October 19, 1972. Following the social hour, dinner was served to 50 members and guests. The business meeting was opened by the President, Dr. Francis J. O'Connor and the minutes of the last meeting were read and accepted. The name of Dr. David A. Beebe of Waterville was read as a new applicant for membership and referred to the Council and for action at the November meeting. Dr. O'Connor announced that the possibility of forming a joint County Bar Association-Medical Association Malpractice Panel would be the subject of our November meeting – to be held in Waterville with the Kennebec County Bar Association.

The Scientific Program was given by Dr. David H. Spodick, Chief, Cardiology Service, Lemuel Shattuck Hospital, Boston, Massachusetts. Dr. Spodick discussed "Medical and Surgical Treatment of Ischemic Heart Disease – What do we know about it?" Dr. Spodick presented a thorough review of the medical aspects of his subject and the known results of surgical approach to coronary artery disease to date. In closing, Dr. Spodick urged objectively designed, controlled trials of therapy in these diseases, whether the methods involved be medical or surgical.

The Kennebec County Medical Association met at the Silent Woman, Waterville, Maine on Thursday, November 16, 1972 in a joint session with the Kennebec County Bar Association. Following the social hour, the business meeting was called to order by the President, Dr. Francis J. O'Connor. The minutes of the last meeting were read and accepted. Dr. David S. Beebe of Waterville was elected to full membership. Dr. Radomsky of the Augusta State Hospital then outlined the purposes of and described the facilities of, the newly opened, nearby, Fairfield Institute, dedicated to the treatment of emotional disorders and alcoholism.

Mr. Sumner H. Lipman, President of the Bar Association then introduced the evening's program which was devoted to discussion of a possible joint Medical Association-Bar Association sponsored Medical Malpractice Panel or plan for medical malpractice cases. The Joint Medical-Legal Plan for such cases currently in use in Androscoggin County was utilized as a sample for discussion and copies of this plan were passed out to the audience. Mr. William Neihoff of the Bar Association discussed details of this proposal. After a thorough question and answer period, it was agreed that our President would appoint a committee to work with the Bar Association in exploring the desirability of a similar plan for Kennebec County. Dr. John W. Towne of Waterville volunteered to serve on this committee.

The meeting was adjourned by Dr. O'Connor at 9:20 p.m.

The annual meeting of the Kennebec County Medical Association was held on Thursday, December 14, 1972 at the Augusta State Hospital. A delicious buffet dinner was served and greetings were extended to the Association by Dr. Ulrich B. Jacobson on behalf of the Augusta State Hospital Staff. Vice-President

Dr. Richard B. Dole presided at the business meeting. The minutes of the last meeting and the Treasurer's Report were read by the Secretary and accepted. The Chairman of the Nominating Committee, Dr. Paul H. Pfeiffer, then presented the following slate of nominations for officers, council, delegates and alternate delegates:

President: Dr. Richard R. Dole, Waterville
Vice-President: Dr. William E. Schumacher, Augusta
Secretary-Treasurer: Dr. Kevin Hill, Waterville
Council: Drs. Joseph J. Hiebel (1 yr.) Waterville, James C. Hayes (2 yrs.) Augusta and Paul Stucki (3 yrs.) Waterville
Delegates to the M.M.A. House of Delegates: Drs. Richard E. Barron, Winthrop, Earle M. Davis, Raymond E. Culver and Anthony Betts, all of Waterville, George I. Gould, Richmond and Terrance J. Sheehan, Augusta. Alternates: Drs. Charles E. Towne and Antoine A. Atallah, both of Waterville, Howard H. Milliken, Hallowell, Robert L. Shelton and Harry M. K. Peddie, both of Augusta

Report of Nominating Committee was accepted and these officers, councilors, delegates and alternate delegates were duly elected unanimously.

The evening scientific program was given by Dr. Richard L. Grant, Associate Professor of Psychiatry, University of Vermont College of Medicine, who chose as his topic "The Problem-Oriented Record and Psycho-Social Health Care." Dr. Grant's discussion included suggestions for more accurate identification of psycho-social problems in a total health care center. Presentation was recorded by video tape and marked the Augusta State Hospital's first formal use of their new video tape recording equipment. The meeting was adjourned by Dr. Dole at 9:00 p.m.

FRANCIS A. SPELLMAN, M.D., *Secretary*

LINCOLN-SAGADAHOC

The regular monthly meeting of the Lincoln-Sagadahoc County Medical Society was held at The Ledges in Wiscasset, Maine on November 21, 1972.

The meeting was called to order by the President, Dr. Anthony J. Keating at 8:45 p.m. The minutes were read by the Secretary and accepted as read.

The Secretary presented the recommendation of the Board of Censors that this Society accept into active membership Dr. Robert H. Dixon of Bath. The nomination was approved unanimously.

Dr. Keating expressed consternation at a 35% increase in malpractice insurance premiums; the situation was discussed generally by the members, with comments from Dr. George W. Wood, III, President of the M.M.A.

Dr. Keating discussed generally the latest contretempts of Bath Memorial Hospital vis-a-vis the State Department of Health and Welfare.

Dr. Keating then introduced Dr. George Wood who spoke about the organization of the Maine Medical Association and about graduate and postgraduate medical education in this State.

The regular monthly meeting of the Lincoln-Sagadahoc County Medical Society was held on December 19, 1972 at The Ledges in Wiscasset, Maine.

The meeting was called to order at 8:50 p.m. by the Vice-President, Dr. Peter A. Evans. The minutes of the November monthly meeting were read by the secretary and accepted as read.

Dr. Evans called for old business and committee reports, of which there were none. He then called upon Dr. Richard C. Leck, who reported on the meeting of the Executive Committee of the M.M.A. on December 10, including EMCRO organizational grants, possible increases in State dues and reasons therefore, Legislative efforts, and the question of registration of physician's assistants. Dr. Nelson P. Blackburn discussed problems in communication among legislators, physicians, and medical society executives.

Dr. Carl R. Griffin, Jr. moved that this Society go on record as supporting an increase in State dues to underwrite a program of legislative liaison between M.M.A. and the State legislature. The motion was seconded and approved.

Dr. Evans appointed a nominating committee consisting of Drs. Kinder, Griffin and Belknap; they were instructed to bring in a slate of officer candidates in January.

Dr. Evans then introduced Dr. Blackburn of the Program Committee, who introduced the speaker of the evening: Dr. Peter Evans. Dr. Evans expatiated on digitalis intoxication.

GEORGE W. BOSTWICK, M.D., *Secretary*

PENOBSCOT

The November meeting of the Penobscot County Medical Society was held on November 21, 1972 at Sing's Restaurant in Bangor, Maine.

Three Penobscot County physicians were elected to membership in the Society. They are: Drs. Mary Harriet Hedrick Hudson, Leonardo Leonor Leonidas and Imre Emery Vincze.

A program describing the Elizabeth Levinson Developmental Center was presented by Dr. John C. Van Pelt, assisted by Miss Carol Done, Mr. Bruce Libby (the Chief Nurse and the Administrator of the Center respectively) and by Albert Anderson, Ph.D. of the Department of Mental Health. The aim of the Center is to bring its residents to their maximum development of human qualities and return them to the community and the parents.

Dr. John A. Woodcock reported briefly on the successful use of Medical Malpractice Screening Committees elsewhere. A new committee was formed with Dr. Woodcock as chairman to investigate the possibility of forming such a committee here.

A resolution concerning the payment of State funds for chiropractic services was discussed by Dr. Dexter J. Clough, 2nd. Action was deferred pending the distribution of the text of the resolution to the membership.

ROBERT L. BURDICK, M.D., *Secretary*

ANDROSCOGGIN

The annual meeting of the Corporators of the Androscoggin County Medical Association was held on December 21, 1972 at Steckino's and called to order by the President, Dr. Thomas F. Shields at 7:00 p.m., with 43 members present.

The minutes of the September meeting were read and corrected; the secretary noted that he had neglected to include the motion, second and passage of change in the Bylaws previously circulated to the membership. The Bylaws are amended to include a quorum of 15 membership present to conduct the business of the Association.

The secretary read a series of correspondence with Mr. David Williams, Director of Part B Medicare, Union Mutual Insurance Company, Portland, Maine. This correspondence was in reply to a motion of the Association regarding various actions recently taken by Part B. Mr. Williams or his representative will be requested to present to the Association a program concerning new regulations and provisions of Part B, including but limited to the recent HR1 legislation.

The request of Dr. Frederic J. Caron for indefinite suspension of membership in the M.M.A. and A.C.M.S. was accepted with regret.

The applications for membership of Drs. Larry Sannes and Jon Pitman were presented following report of the Credentials Committee. Drs. Sannes and Pitman were admitted to membership.

Standing Committee reports were read and accepted. Following acceptance of the report of the Treasurer and Finance Committee, it was moved and seconded to transfer $4,000.00 in additional funds to the Finance Committee for investment under

the able direction of Drs. Tibbetts, Harkins and Fortier.

A motion was passed instructing the President to appoint a committee to investigate the original plans of the investment fund and submit proposals for the distribution of accumulated funds.

At the request of the Treasurer, it was moved and seconded to increase the annual budget for secretarial services contracted by the Association to $600.00.

The nominating committee reported their proposed plans of new officers and under motion duly seconded a unanimous ballot (Dr. Charles W. Steele, chairman of nominations).

President: Dr. Gilbert R. Grimes, Lewiston

Vice-President: Dr. Gerard L. Morin, Lewiston

Secretary-Treasurer: Dr. Richard M. Swengel, Lewiston

Counselor: Dr. Lawrence A. Nadeau, Lewiston

Delegates to the M.M.A. House of Delegates: Drs. Thomas F. Shields, Behzad Fakhery and Albert Shems, all of Lewiston. Alternates: Drs. John W. Carrier, Lawrence A. Nadeau and Richard A. Marshall, all of Lewiston

Dr. Shields thanked the officers, committee members and membership at large for their assistance during the past year before turning the chair over to Dr. Grimes.

The meeting adjourned at 9:00 p.m.

RICHARD M. SWENGEL, M.D., *Secretary*

Letter to the Editor

To the Editor:

You will recall that the Food and Drug Administration's Bureau of Radiological Health has responsibility for developing and administering a radiation control for health and safety program as authorized by Public Law 90-602. Under that program, a radiation safety performance standard for diagnostic x-ray equipment was published on August 15, 1972, to become effective one year later.

The Bureau recently learned that some dealers have been advising physicians and other users that all existing x-ray equipment will have to be upgraded to meet requirements of the standard by the effective date of August 15, 1973. You may be able to perform a service for your readers by informing them that such advice is contrary to fact.

Upgrading of x-ray equipment now being used is not now required by the standard. State and territorial radiation control authorities have been asked by the Bureau to so inform equipment users and dealers.

Our communication to the States and territories made one other point. This was that, although equipment now in use will not have to be modified before the standard becomes effective, owners installing manufacturer-certified components in such x-ray systems after next August 15 must install components of the type called for by the Federal standard.

Additional information about the standard may be obtained from the Division of Electronic Products, Bureau of Radiological Health, Food and Drug Administration, 12720 Twinbrook Parkway, Rockville, Maryland 20852.

JOHN C. VILLFORTH
Director
Bureau of Radiological Health

PROCAINAMIDE AND LUPUS ERYTHEMATOSUS — *Continued from Page* 55

ANAs, ranging from 1/4 to 1/128.[2] Then I would repeat this at three months and at six months as it has been observed[5] that most changes occur in the first four months. I would then obtain ANAs every six months or whenever suggestive symptoms, especially arthralgias appear. The one problem that is not clearly defined in the literature is that of a rising titre. It would seem wise to use an alternative drug, if possible, if the titre rises over 1/64.

REFERENCES

1. Martin, Eric W., Ph.D., Editor: Hazards of Medication. J. B. Lippincott Company, Philadelphia and Toronto: 301, 1971.
2. Svec, Kathryn H. and Veit, Bruce C.: "Age-Related Antinuclear Factors: Immunologic Characteristics and Associated Clinical Aspects." Arthritis and Rheumatism. 10; 6: 509-516, 1967.
3. Fakhro, Ail M., Ritchie, Robert F., Lown, Bernard: "Lupus-like Syndrome Induced by Procainamide." American Journal of Cardiology. 20; 367-373, 1967.
4. Molina, Javier, Dubois, Edmund L., Bilitch, Michael, Bland, Stephen L., Friou, George J.: "Procainamide-Induced Serologic Changes in Asymptomatic Patients." Arthritis and Rheumatism. 12; 6: 608-614, 1969.
5. Blomgren, Stephen E., Condemi, John J., Bignall, Margaret C., Vaughan, John H.: "Antinuclear Antibody Induced by Procainamide." New England Journal of Medicine; 281: 64-66, 1969.
6. Russell, A. S. and Ziff, M.: "Natural Antibodies to Procaine Amide." Clinical Immunology; 3: 901-909, 1968.
7. Byrd, Richard B. and Schanzer, Bernard. "Pulmonary Sequelae in Procaine Amide Induced Lupus-like Syndrome." Diseases of the Chest; 55, 2: 170-172, 1969.
8. Alarcon-Segovia, Donato: "Drug-Induced Lupus Syndromes." Mayo Clinic Proceedings; 44: 664-681, 1969.
9. Holley, Howard L.: "Evidence for a Predisposition to Rheumatic Diseases in Families of Patients Developing Drug-Induced Systemic Lupus Erythematosus." Arthritis and Rheumatism; 7: 6, 684-686, 1964.
10. Sheldon, P. J. H. S. and Williams, W. R.: "Procainamide-induced Systemic Lupus Erythematosus." Annals of Rheumatic Diseases; 29: 236-243, 1970.
11. Lappat, E. J. and Cawein, M. J.: "A Familial Study of Procainamide-Induced Systemic Lupus Erythematosus." American Journal of Medicine; 45: 846-852, 1968.
12. Dubois, Edmund L.: "Procainamide Induction of a Systemic Lupus Erythematosus-Like Syndrome." Medicine; 48, 3: 217-228, 1969.
13. Perry, Jr., H. Mitchell, Tan, Eng M., Carmody, Sharon, Salamoto, Arthur: "Relationship of Acetyl Transferase Activity to Antinuclear Antibodies and Toxic Symptoms in Hypertensive Patients Treated with Hydralazine." Journal of Laboratory and Clinical Medicine; 76: 114-125, 1970.
14. Kendall, M. J., Hawkins, C. F.: "Quinidine-Induced Systemic Lupus Erythematosus." Postgraduate Medical Journal; 46: 729-731, 1970.

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Primary Mesenteric Infarction

W. Edward Jordan, Jr., M.D.

Introduction

Primary mesenteric infarction is an uncommon, but highly fatal disease which presents a challenge to the management and diagnostic acumen of the attending surgeon. Overall survival rates reported in the literature are quite low.[1,2] At one time, it was felt that vascular procedures performed on mesenteric vessels would cause a marked increase in survival. This has not proved to be the case.[1] The mainstays of successful management remain the treatment of shock, diagnosis, and resection of infarcted gut. Three cases of this condition have been treated by the author at the Redington-Fairview General Hospital in the past two years. A review of the literature and study of these cases should be instructive.

Review of the Literature

Slater and Elliott[2] from Pittsburgh presented 18 cases seen in a 10-year period. They classify the condition into four groups:

1. Arterial thrombosis
2. Venous thrombosis
3. Embolism
4. Non-occlusive

The incidence was low, but the mortality rate was high. Only one case survived, although at postmortem examination, 9 of the 18 had resectable areas of infarcted gut.

Ottenger and Austin[1] discuss 136 cases with 11 survivors. They report the incidence as 8.8 per 100,000 admissions to the Massachusetts General Hospital. Sixty-seven of their patients fell into the non-occlusive group of Slater and Elliott.

Marston[3] states that most patients who die of mesenteric infarction had a prolonged period of intestinal angina preceding infarction. Intestinal angina is characterized by pain, frequently after meals, aggravated by exercise, and lasting about one hour. There is weight loss and absence of other gastrointestinal pathology in the arteriosclerotic individual. It should be remembered that due to collateral circulation complete vascular occlusion can be present without the syndrome.

Williams[4] discusses 45 cases of mesenteric infarction, 80% of which presented no major vessel occlusion. By the time bleeding, peritonitis, and shock were present, the diagnosis was obvious and survival was extremely low. He feels the first 30 hours are most important and that early arteriography is helpful and recommends a second look operation.

The appearance of severe, steady pain may herald strangulated bowel. The patient looks ill, has fever, and rebound tenderness. Dark bloody fluid can be aspirated from the peritoneal cavity, and stools frequently contain blood.[5] Temperature and white blood cell count are elevated. At this stage there is a severe fluid deficit. Helpful modalities mentioned in the literature include Solu-Medral® to combat endotoxic shock and irrigation of the peritoneal cavity with cephalosporius.

Presentation of Three Cases

Summary of Case #1

A 47-year-old white female was admitted to the Redington-Fairview General Hospital on the 27th of February 1970.

Problem #1: Abdominal pain upgraded at laparotomy to mesenteric thrombosis.

SUBJECTIVE: This 47-year-old woman became ill one month prior to admission with generalized abdominal pain. She was hospitalized at the Jackman, Maine facility. Her condition became progressively worse – vomiting and diarrhea developed – the pain became constant and unrelenting and she was transferred to the Redington-Fairview General Hospital.

OBJECTIVE: There was a healed right upper quadrant scar. The lower abdomen was distended. Bowel sounds were present and high-pitched. The abdomen was generally tender. WBC was 14,800. A flat film revealed small bowel gas.

THERAPY: Following restoration of fluid balance with vigorous intravenous therapy, the patient was explored on the 1st of March and mesenteric thrombosis was discovered and 150 cm. of bowel was resected with the path report of venous mesenteric thrombosis. She recovered after a stormy course and was discharged on April 24th.

Problem #2: Right lower quadrant abscess.

SUBJECTIVE: Spiking fever and pain in the right lower quadrant developed on the 19th postoperative day.

OBJECTIVE: There was dullness to percussion over a large area in the right lower quadrant. Over the next 24 hours, there were signs of increased accumulation of fluid.

THERAPY: Heparin was discontinued and the patient was operated on the 20th of March with drainage of a large right lower quadrant abscess.

Problem #3: Pelvic abscess.

SUBJECTIVE: Malaise.

OBJECTIVE: On the 30th postoperative day the patient developed increasing fever and rise in the white count. Bi-manual examination revealed a large mass which filled the pelvis.

THERAPY: Antibiotics were administered. The mass became fluctuant and drained spontaneously into the area of the drains in the right lower quadrant.

Problem #4: Wound disruption.

SUBJECTIVE: Massive drainage.

OBJECTIVE: There was massive drainage of bloody fluid from the wound which occurred on the fifth postoperative day. Inspection revealed that the wound had dehisced. Wire sutures and peritoneum were exposed, but intestine did not protrude.

THERAPY: The patient at this stage was too sick to return to the operating room. A vaginal pack was soaked with Furacin® solution and placed into the wound and a tight dressing applied. Secondary closure of this wound was performed on the 20th of March, 15 days after the dehiscence and under the same anesthesia under which the right lower quadrant abscess was drained. It healed uneventfully thereafter.

Problem #5: Diabetes Mellitus.

SUBJECTIVE: Zero.

OBJECTIVE: A blood sugar of 300 and a 4+ urine sugar and acetone were present shortly after admission. This was regulated with regular Insulin during the first 24 hours, 70 units being required. Thereafter, 30-40 units of Insulin were required daily throughout her hospital stay. She was placed on Orinase® prior to discharge.

Problem #6: Anti-coagulant therapy.

SUBJECTIVE: Zero.

OBJECTIVE: Heparin was administered, in a dose of 50-75 mg. subcutaneously at 6 hour intervals. Lee White times were maintained between 15 and 25 minutes as determined 1 hour after the dose. This medication was discontinued for the operative procedure mentioned above.

The patient was discharged from the hospital on the 56th hospital day taking Orinase® and a 1200 calorie diabetic diet.

Summary of Case #2

A 50-year-old male was admitted to the hospital on December 28, 1970.

Problem #1: Abdominal pain upgraded at laparotomy to mesenteric thrombosis.

SUBJECTIVE: 24 hours prior to admission the patient developed the sudden onset of abdominal pain, severe, unrelenting and associated with nausea and vomiting.

OBJECTIVE: Acutely ill white male with rapid pulse, generalized abdominal tenderness and rebound tenderness. The WBC rose progressively to a level of 31,000. Flat abdominal film showed small bowel gas.

THERAPY: He was seen in surgical consultation on the 6th day of his hospital stay and after restoration of fluid and electrolyte balance was explored the same evening with resection of 35 cm. of gangrenous bowel. The path report was infarct of small intestine, venous. Gradual recovery ensued after a long illness.

Problem #2: Dehydration.

SUBJECTIVE: Restless and complaining of dryness of the mouth. The patient was uncooperative.

OBJECTIVE: The urine specific gravity was 1022, output was scanty being 240 cc. on the day prior to surgery. Mild hypokalemia was present with a serum value of 3.6 mg.%.

THERAPY: Electrolyte and potassium chloride solutions sufficient to obtain an output of 30-40 cc. per hours and a normal serum electrolyte pattern were administered; 4,000 cc. being required in the first 24 hours. Postoperatively fluid and electrolyte balance were maintained by replacement of measured and insensible loss.

Problem #3: Right lower lobe atelectasis.

SUBJECTIVE: On the 15th hospital day, the patient developed a secondary temperature rise to 103 Fahrenheit which persisted for four days.

OBJECTIVE: The lungs were clear. The chest showed linear atelectasis on the right base on chest x-ray.

THERAPY: Antibiotics and coughing regime resulted in subsidence of the symptomatology.

Problem #4: Anti-coagulant therapy.

SUBJECTIVE: Zero.

OBJECTIVE: Subcutaneous Heparin was given every 6 hours, 50 mg. and Lee White times were maintained between 15 and 30 minutes as measured 1 hour after administration of Heparin.

Problem #5: Upper GI bleeding.

SUBJECTIVE: The patient felt nauseous and faint and vomited black material on the 20th postoperative day.

OBJECTIVE: Blood pressure fell to 86/40. The Lee White time was 21 minutes. Hemoglobin after the bleed was over and the loss had been diluted was 7.6 grams. Follow-up GI series revealed a duodenal diverticulum.

THERAPY: 500 cc. of whole blood were given with restoration of the vital signs.

The patient was discharged on the 33rd postoperative day.

Summary of Case #3

This 74-year-old white female was admitted to the hospital on the 7th of January 1972.

Problem #1: Abdominal pain upgraded at laparotomy to mesenteric thrombosis.

SUBJECTIVE: The patient developed severe, unrelenting pain 8 hours prior to admission with associated nausea and vomiting.

OBJECTIVE: Generalized tenderness and rebound tenderness, maximum in the right upper quadrant. The white blood count was 14,700. Abdominal flat film showed a moderate amount of small bowel gas.

THERAPY: Exploration on the 8th of January was performed. Resection of 90 cm. of gangrenous bowel was accomplished. A stormy postoperative course followed with death 48 hours later. Postmortem findings of recurrent mesenteric thrombosis.

Problem #2: Dehydration.

SUBJECTIVE: Complaints of thirst.

OBJECTIVE: Skin and mucous membranes were dry. The skin turgor was poor. Urine output was low.

THERAPY: 3,000 cc. of electrolyte solution brought the urine output to a level of 10-20 cc. per hour with a central venous pressure of 9. Attempts to increase fluid administration above this level resulted in rapidly rising central venous pressure.

COURSE: Urine output fell to the vanishing point prior to death.

Problem #3: Diabetes.

SUBJECTIVE: Zero.

OBJECTIVE: The blood sugar was 190 on admission. 25 units of Insulin were given during the first 24 hours to cover the glucose given by intravenous administration and the blood sugar was thereafter not a problem.

Problem #4: Cardiac decompensation with digitalis toxicity.

SUBJECTIVE: This patient takes digitalis regularly and was complaining of nausea.

OBJECTIVE: The pulse rate was slow. Electrocardiogram showed an aterial fibrillation with multiple PVC's.

THERAPY: Fluids were given cautiously. Digitalis was withheld. Lasix® was without effect.

Problem #5: Obstructive bronchopulmonary disease.

SUBJECTIVE: Complaints of wheezing respirations.

OBJECTIVE: The chest was emphysematous. Chest x-ray

revealed infiltration at the right base. Preoperatively breathing room air, pH 7.47; PCO2, 32; and PO2, 41. Postoperatively on respirator support, the pH was 7.38; PCO2, 50; and PO2, 40. During induction of anesthesia, there was an episode of aspiration.

COURSE: The patient ran a progressive downhill course and died 48 hours postoperatively. Postmortem findings of aspiration pneumonitis were present.

Discussion and Conclusion

The cases presented illustrate many of the problems involved in the management of primary mesenteric infarction. All three patients were functionally active at home at the time of the onset of their condition. Only one patient (Mrs. T. C.) had a history which could have represented intestinal angina. No one of the three had a really major vessel occlusion. Initial impression in one patient included mesenteric thrombosis, but in spite of this three days went by prior to exploration. Only one patient had significant cardiac disease. Bowel sounds were present in two and in one of these they disappeared under observation. Marked and increasing elevation of the white blood cell count was the rule, with shift of the differential to the left. Abdominal films were not particularly helpful but in retrospect showed an increase in overall density and small bowel gas. Anti-coagulants were used postoperatively in the two survivors.

This disease is universally fatal if untreated surgically, and it would seem from findings in the literature that approximately ½ of these patients have resectable gut. Vigorous surgical intervention is certainly indicated. It is not surprising that shock is often less after the operation than it was before. Dehydration occurs quickly out of proportion to the external losses because of the accumulation of peritoneal fluid. This dehydration masks a considerable loss of blood. Vigorous preoperative fluid therapy, sufficient to produce a good urine output, is needed. This is thrombo-embolic disease and it is my feeling that anticoagulants should be employed.

Acknowledgment

This work was supported in part by a grant from the Bio-Medical Institute.

References

1. Study of 136 Patients with Mesenteric Infarction, Ottinger & Austin, S. G. & O., Feb. 1967.
2. Primary Mesenteric Infarction, Slater & Elliott, A., J. of Surg., March, 1972.
3. Mesenteric Arterial Disease, Marston, Gut 1967.
4. Ischemic Disease of Small Bowel, Williams, et al, Disease of Colon and Rectum, July-August, 1970.
5. Critical Surgical Illness, Hardy, Saunders 1971, pg. 477-488.

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Congenital Anomalies of the Gallbladder

CARLTON E. SWETT, M.D., F.A.C.S.

Congenital anomalies of the gallbladder constitute a relatively uncommon condition in man, and, therefore, infrequently encountered by the general surgeon. This condition is easily missed preoperatively and can be as easily missed during surgery on the biliary tree if it is not suspected. X-ray studies of the biliary tree may not visualize both gallbladders or may demonstrate the normal organ and not the diseased one. This paper will present a brief review of the recent literature and present two cases of congenital anomalies of the gallbladder.

CASE REPORTS

Case #1: This 28-year-old multiparous white female was admitted to the Fairview Hospital in October 1955, complaining of recurrent right upper quadrant pain radiating to the back. It was suspected at that time that she had gallbladder disease and x-rays revealed multiple gallstones. She was hospitalized in November 1955, and underwent exploratory laparotomy and cholecystectomy and appendectomy. Pathology report revealed two gallstones and microscopic examination revealed chronic cholecystitis. The appendix was not remarkable. There is nothing in the operative report to indicate that the surgeon suspected an anomaly of the biliary system. The patient had an uneventful recovery and was well for approximately nine years when she was again hospitalized at the same institution for evaluation of recurrent, severe right upper quadrant pain. Diagnostic studies at that time revealed pancreatitis. Upper gastrointestinal series and electrocardiogram were normal. The patient was seen in consultation by the author who felt that the patient had pancreatitis as the result of recurrent or retained common duct stones. The patient was returned to the operating room on February 5, 1964, and exploratory laparotomy, lysis of extensive adhesions, operative cholangiogram and common duct exploration were carried out. Several common duct stones were found and the ampulla of Vater was found to be moderately stenosed and was dilated. Operative cholangiogram prior to exploration of the common duct revealed free flow of the dye into the duodenum and no abnormalities were noted other than the stones within the common duct. The common duct was closed in the usual fashion over a T-tube. On the 10th postoperative day, a T-tube cholangiogram was performed prior to discharging the patient from the hospital and this revealed a normal common duct; however, a cystic duct connecting to a partially filled gallbladder containing multiple radiolucent stones was identified. The patient was informed of the congenital abnormality with the presence of a second gallbladder. She was re-admited to the same hospital on March 2, 1964, and underwent laparotomy, choledochotomy, cholecystectomy, and operative cholangiography. This operative procedure was prolonged because the second gallbladder was difficult to locate. It was finally located to the right of the common duct in a superior and posterior position to the first portion of the duodenum. Cholecystectomy was performed without any problems after the organ had been identified. The gallbladder contained multiple stones and pathologically revealed chronic cholecystitis and cholesterolosis. Recovery from this operation was uncomplicated and the patient has had no further difficulty since that time.

Case #2: This 23-year-old multiparous female was seen by her physician because of persistent upper abdominal pain. Upper gastrointestinal series and gallbladder x-rays were performed, revealing a normal upper gastrointestinal series and a congenital malformation of the gallbladder without evidence of stones. The x-rays revealed what appeared to be a bifid gallbladder. Because of the absence of stones, the patient was treated medically, but her symptoms failed to improve. She was hospitalized for further investigation and surgical consultation on November 8, 1972. Physical examination revealed a healthy appearing lady with entirely normal physical findings. Abdominal examination was normal, and there was a well healed appendectomy scar. Pelvic examination was normal. Basic laboratory studies were all within normal limits. After three months of medical treatment her symptoms were not improved and it was felt that removal of the malformed gallbladder was indicated. The patient underwent exploratory laparotomy, cholecystectomy and operative cholangiography on November 10, 1972. Pathologic examination revealed a bifid gallbladder. The postoperative convalescence was without complications and the patient's symptoms have been relieved.

DISCUSSION

Congenital anomalies of the gallbladder may present in several different ways. The two case presentations describe two types of congenital anomalies of the gallbladder. True double gallbladders, as illustrated by Case #1, are two distinctly separate gallbladders, each having its own cystic duct entering the hepatic or common bile duct. They may or may not have a common peritoneal covering. Bifid gallbladder, or bilobed gallbladders, as illustrated by the second case presentations, are less common and are a result of an abnormal or incomplete formation of the gallbladder embryologically. The gallbladder is formed by a budding outgrowth of the bile duct. If more than one of these gallbladder buds persist, then multiple gallbladders will be the result. Case #1 represents complete development of two embryologic buds of the common duct.

The incidence of anomalies of the gallbladder is difficult to determine. Palmisano reported in 1959 that there were fifty-two cases of double gallbladder in the world literature to that date. Hobby in the *British Journal of Surgery* in 1970 reported twelve cases of bilobed gallbladder to that date. This would indicate that the incidence of double gallbladder is greater than that of the bilobed, or septate gallbladder.

SUMMARY

Case reports of two distinct types of gallbladder anomalies have been presented. The first case was that of a true double gallbladder in which there were two separate gallbladders and two cystic ducts. The second case was that of a bilobed gallbladder where there is a septum dividing the body of the gallbladder but the gallbladder enters the common duct via a common cystic duct. The literature had been briefly reviewed indicating this is an

Continued on Page 78

Cardiopulmonary Complications in the Surgical Patient

John R. Hewson, M.D.*

Synopsis – Abstract

Detailed analysis of 616 consecutive surgical procedures showed angina pectoris to increase the risk of postoperative myocardial infarction 22-fold. Preoperative congestive heart failure predisposed to pneumonia, congestive heart failure, and respiratory insufficiency postoperatively. Asthma increased the risk of respiratory insufficiency 15-fold. Preoperative pneumonia was associated with an increased risk of congestive heart failure, cardiac arrhythmias, respiratory insufficiency, and death. Cardiac arrhythmias prior to surgery predisposed to life-threatening arrhythmias postoperatively, and apparently also to renal failure. Chronic bronchitis and emphysema each led to increased risk of congestive heart failure and pneumonia, and emphysema also to respiratory insufficiency postoperatively. Previous remote myocardial infarction did not increase surgical risk. The type of anesthesia was likewise not a significant factor. "Central" surgery carried a greater risk of congestive heart failure and cardiac arrhythmias.

As surgical expertise increases, and the life expectancy of our population lengthens, the medical aspects of the surgical patient play a progressively greater role in the ultimate outcome of surgical intervention.

This paper presents an analysis of the preoperative status and postoperative complications and their interrelationships in all surgical cases over a one-year period in a community hospital.

Materials and Methods

All cases coming to surgery in a 60-bed community hospital over a twelve-month period were analyzed. The author performed none of the surgery. There were 616 surgical procedures in all. Each chart was completely reviewed by the author, so that assessment of each case was based on all details in the chart, not just the discharge diagnosis.

Each clinical entity was defined according to usual criteria. Such an entity was also considered to be present preoperatively if medical therapy was necessary to prevent its clinical manifestation. Postoperative complications were defined by the usual appropriate clinical and/or laboratory or pathological criteria. Each entity was entered on punch cards, one for each surgical procedure performed.

GENERAL: Age; sex.

PREOPERATIVE PATHOLOGY: Angina pectoris; previous myocardial infarction; congestive heart failure; chronic bronchitis; emphysema; asthma; preoperative pneumonia; remote pulmonary embolus; cerebrovascular disease.

INTRAOPERATIVE FACTORS:

(1) *Site of Surgery*

[a] *Peripheral* – all limb surgery, rectal surgery, ophthalmological surgery, oral procedures including tonsillectomy, and cystoscopy with or without retrograde pyelography.

[b] *Low abdominal surgery* – all gynecological surgery including Caesarian section, simple appendectomy, inguinal, femoral and ventral herniorrhaphy, suprapubic prostatectomy and suprapubic cystotomy.

[c] *Central surgery* – all standard major gastrointestinal surgery (excluding simple appendectomy), nephrectomy, radical mastectomy, thyroidectomy, esophagoscopy, and bronchoscopy.

(2) *Type of Anesthesia*

General, spinal, epidural, local and regional block.

POSTOPERATIVE COMPLICATIONS: Angina pectoris; acute myocardial infarction; congestive heart failure; cardiac arrhythmia (this included patients who suddenly died with no apparent explanation); pneumonia; pulmonary embolus; respiratory insufficiency; hepatic failure; stress ulcer; acute cerebrovascular accident; wound infection; septicemia; renal failure; shock; death.

Results

CORRELATION OF POSTOPERATIVE COMPLICATIONS WITH PREOPERATIVE PATHOLOGY (TABLE 1)

Preoperative angina pectoris was associated with a 6.5 percent incidence of proven postoperative myocardial infarction, a 22-fold increase in the occurrence or risk of acute postoperative myocardial infarction.

Previous myocardial infarction, by itself, was not associated with any significantly increased risk of postoperative complications.

Preoperative congestive heart failure proved dangerous, with a 10 to 20 percent occurrence of postoperative congestive heart failure, pneumonia, and respiratory insufficiency, a 4- to 9-fold increase.

Preoperative cardiac arrhythmias were associated with a 13- and 9-fold increase in serious postoperative cardiac arrhythmias and renal failure, 13.5 and 9.0 percent respectively.

*Chief of Staff, Maine Coast Memorial Hospital, Ellsworth, Maine 04605.

TABLE 1

CORRELATION OF POSTOPERATIVE COMPLICATIONS WITH PREOPERATIVE PATHOLOGY

Preoperative Pathology (PP)	*Associated Postop Complic (PC)*	*Minimum Age Years*	*Incidence of Postop Complication (PC)*				*Increase in Risk of PC with PP*	*Statistical Significance (P <)*
			Number in Patients		*Percent in Patients*			
			With PP	*Without PP*	*With PP*	*Without PP*		
ANGINA	ACUTE M I	41	2/31	1/325	6.5%	0.3%	22	0.05
C H F	C H F	51	4/35	4/246	11.4%	1.6%	7	0.01
	PNEUMONIA	51	6/35	10/246	17.1%	4.1%	4	0.01
	RESP INSUFF	51	4/35	3/246	11.4%	1.2%	9	0.01
ARRHYTHMIA	ARRHYTHMIA	31	3/22	4/394	13.5%	1.0%	13	0.001
	RENAL FAILURE	31	2/22	4/394	9.0%	1.0%	9	0.05
CHR BR	C H F	41	4/23	4/333	17.4%	1.2%	14	0.001
	PNEUMONIA	41	5/23	12/333	21.7%	3.6%	6	0.001
EMPHYSEMA	C H F	41	4/29	4/327	13.8%	1.2%	11	0.001
	PNEUMONIA	41	6/29	11/327	20.7%	3.4%	6	0.001
	RESP INSUFF	41	3/29	5/327	10.3%	1.5%	7	0.05
ASTHMA	RESP INSUFF	11	2/11	6/559	18.2%	1.1%	16	0.001
PNEUMONIA	C H F	21	2/10	6/494	20.0%	1.2%	17	0.001
	ARRHYTHMIA	21	2/10	5/494	20.0%	1.0%	20	0.001
	RESP INSUFF	21	3/10	5/494	30.0%	1.0%	30	0.001
	DEATH	21	2/10	15/494	20.0%	3.0%	7	0.05
C V D	SEPTICEMIA	51	2/18	3/263	11.1%	1.1%	10	0.05
	DEATH	51	4/18	13/263	22.2%	4.9%	5	0.05

ACUTE M I = acute myocardial infarction
C H F = congestive heart failure
CHR BR = chronic bronchitis
C V D = cerebrovascular disease
RESP INSUFF = respiratory insufficiency

TABLE 2

CORRELATION OF POSTOPERATIVE COMPLICATIONS WITH SITE OF SURGERY

Non-Peripheral Surgery (NPS)	*Postoperative Complication (PC)*	*Minimum Age Years*	*Incidence of Postoperative Complication*				*Increase in Risk of PC With NPS*	*Statistical Significance (P <)*
			Number in Patients		*Percent in Patients*			
			With NPS	*With PS*	*With NPS*	*With PS*		
CENTRAL	C H F	61	7/64	1/104	10.9%	1.0%	11	0.01
	ARRHYTHMIA	61	6/64	0/104	9.4%	0.0%	>9	0.01
	HEPAT FAIL	61	4/64	0/104	6.2%	0.0%	>6	0.05
	WOUND INFECT	11	11/111	1/223	9.9%	0.4%	25	0.001
LOW ABD	WOUND INFECT	11	19/236	1/223	8.0%	0.4%	20	0.001

C H F = congestive heart failure
HEPAT FAIL = hepatic failure
LOW ABD = low abdominal
PS = peripheral surgery
WOUND INFECT = wound infection

Both chronic bronchitis and emphysema were associated with a 14 to 22 percent incidence of postoperative congestive heart failure and postoperative pneumonia, a 6 to 14 times increase in risk. Emphysema, unlike chronic bronchitis, however, had a 10 percent postoperative incidence of the serious problem of respiratory insufficiency, a 7-fold increase in incidence.

Asthma proved to be equally dangerous. Almost one-fifth developed severe respiratory insufficiency postoperatively, a 16-fold increase in incidence of this life-threatening complication.

Preoperative pneumonia was also very dangerous. Postoperative congestive heart failure, cardiac arrhythmia, respiratory insufficiency, and death, each had an incidence of 20 to 30 percent in cases with this preoperative pathology, an increase of 7 to 30 times in the occurrence of these postoperative problems.

Preoperative cerebrovascular disease was associated with an approximate 10 to 20 percent incidence of septicemia and death, a 10- and 5-fold increase in incidence, respectively.

CORRELATION OF POSTOPERATIVE COMPLICATIONS WITH SITE OF SURGERY (TABLE 2)

In cases with "central" surgery, approximately 10 percent developed congestive heart failure and cardiac arrhythmia, an increase of 10 times, compared to peripheral surgery. Postoperative liver failure and wound infection were 6 to 25 times greater in "central" than in peripheral surgery, but the nature of the surgery itself (colonic resections, biliary surgery), could explain this difference.

Table 3

Correlation of Preoperative Pathology and Postoperative Complications With Type of Anesthesia, Compared With General Anesthesia

Type of Non Gen Anes (NGA)	Category of Clinical Parameter	Specific Clinical Parameter	Age Group Years	Incidence of Clinical Parameter: Number in Patients With NGA	Number in Patients With GA	Percent in Patients With NGA	Percent in Patients With GA	Factor Increase With NGA	Statistical Significance (P <)
EPIDURAL	PREOP	PNEUMONIA	>21	5/115	1/315	4.3%	0.3%	14	0.01
		PR P EMB	>31	3/90	1/254	3.3%	0.4%	8	0.05
	POSTOP	WND INFEC	>11	13/120	17/373	10.8%	4.6%	2	0.01
SPINAL	PREOP	PREV M I	>41	9/29	23/209	31.0%	11.0%	3	0.01
	POSTOP	HEPAT F	>61	2/27	1/115	7.4%	0.9%	8	0.05
BLOCK	PREOP	PNEUMONIA	>21	2/20	1/315	10.0%	0.3%	33	0.01
LOCAL	PREOP	ANGINA	>41	6/24	17/209	25.0%	8.1%	3	0.05
		PNEUMONIA	>21	2/25	1/315	8.0%	0.3%	27	0.01
	POSTOP	SEPTICEMIA	>31	2/24	1/254	8.3%	0.4%	21	0.05

GA = general anesthesia
HEPAT F = hepatic failure
NGA = type of anesthesia other than general inhalational anesthesia
PREV M I = previous myocardial infarction
PR P EMB = previous pulmonary embolus
WND INFEC = wound infection

CORRELATION OF POSTOPERATIVE COMPLICATIONS WITH TYPE OF ANESTHESIA (TABLE 3)

No significant differences in cardiopulmonary complication rates were found among the various types of anesthesia. There was an 8-fold increase in incidence of liver failure with spinal anesthesia, a 21-fold increase in septicemia with local anesthesia, and a 2-fold increase in the wound infection rate with epidural anesthesia.

Discussion

In the present study, angina pectoris had a 22-fold increase in incidence of acute postoperative myocardial infarction, an incidence of 6.5 percent. Two other studies have reported 0.0 percent[1] and 10.8 percent[2] occurrence of acute postoperative myocardial infarction in patients with angina pectoris. The mortality rate in those with angina pectoris who undergo surgery was 9.7 percent in the present study. As far back as 1930, a similar mortality rate of 12.2 percent was reported for angina patients.[3] Values in more recent series range from 0 to 16.2 percent.[1,2,4] It would appear that over the past 40 years the surgical risk posed by the existence of angina pectoris has not altered to an appreciable extent. Of those in the present study who did develop an acute postoperative myocardial infarction, 66.7 percent died. This horrendous mortality is similar to other reports in the literature, with no evidence of much improvement over the years.[3,5,6,7,8] In the light of the high risk of postoperative myocardial infarction in patients with angina, it has been suggested that they have intensive physiological monitoring both intra- and postoperatively, if this complication and its attendant mortality are to be diminished.[9]

The present study failed to reveal any significant increase in complication rates in patients who had suffered a previous myocardial infarction. Many of these previous infarctions could not be dated, but those in which the time was known, had occurred more than three years previously. Knapp in 1962[8] likewise found no postoperative myocardial infarction in patients whose previous infarction had occurred more than 3 years before, but a 100 percent incidence in those with infarcts within the preceding 6 months. Others have similarly failed to show any high degree of attendant cardiac or pulmonary complications with remote myocardial infarction.[1,2]

Preoperative congestive heart failure had a 4 to 9 times greater incidence of postoperative congestive heart failure, pneumonia, and respiratory insufficiency. Left ventricular failure results in pulmonary impairment,[10–13] which could explain the respiratory insufficiency found in the present study, as well as the propensity for pneumonia. Pulmonary edema has recently been shown to cause severe acute respiratory acidosis.[14–17]

There was a 13-fold increase in the incidence of postoperative cardiac arrhythmias in patients with preoperative cardiac arrhythmias in the present study. Eighty-five percent of those with postoperative arrhythmias died, emphasizing the danger of preoperative and postoperative arrhythmias. Intraoperative cardiac arrhythmias occur with higher incidence in patients with preoperative arrhythmias.[18] Spontaneous ventilation during general anesthesia has been found to be associated with an increased incidence of intraoperative cardiac arrhythmias.[19,20] Intraoperative respiratory acidosis is a common cause of cardiac arrhythmias with cyclopropane[21–24] and with halothane.[25] Even inhalation of carbon dioxide in unanesthetized subjects can cause electrocardiographic abnormalities.[26] In a study of cardiac arrests, half were found to be in respiratory acidosis just before the arrest, and one quarter were in metabolic acidosis with compensatory hypocarbia.[27] It is postulated that acidosis causes catecholamine release through stimulation of the vasomotor center of the brain stem, causing cardiac arrhythmias.[18] It is well documented that hypoxia can lead to release of catecholamines and to cardiac arrhythmias.[28–36] The occurrence of renal failure in patients with preopera-

tive cardiac arrhythmias in the present study raises the possibility of the existence of poor renal perfusion secondary to the arrhythmia.

The patient with chronic obstructive lung disease has a much increased risk of developing postoperative pneumonia and also congestive heart failure. Those with emphysema were also found to have an increased risk of respiratory insufficiency, while those with bronchitis without evidence of emphysema did not. Hypoxia and hypercapnea have each been shown to greatly diminish renal function, causing fluid retention which clinically is indistinguishable from cardiac failure.[37] The findings in the present paper may therefore be due to a renal rather than a cardiac response to altered pulmonary function. Stein has demonstrated that chest physiotherapy does reduce pulmonary complications in surgical patients with chronic obstructive lung disease.[38]

The 18 percent incidence of postoperative respiratory insufficiency in asthmatics in the present paper is quite striking. Asthmatics have bronchial obstruction due to mucosal edema, smooth muscle spasm, and thick tenacious sputum.[39] They have been shown to suffer pulmonary impairment even in the absence of overt symptoms.[40,41] Aggressive pulmonary care is required by the asthmatic surgical patient.[42]

Preoperative pneumonia is highly dangerous, with its propensity to precipitate postoperative congestive heart failure, cardiac arrhythmias, respiratory insufficiency, and death. The congestive heart failure may actually be fluid retention secondary to renal impairment due to hypoxia,[37] and the cardiac arrhythmias could perhaps be due to respiratory acidosis[21–27] or hypoxia.[28–36]

Previous cerebrovascular disease did not significantly predispose to acute postoperative cerebrovascular accident. Others have similarly failed to find any significant predisposition.[8]

The incidence of congestive heart failure and cardiac arrhythmias postoperatively in cases of "central" surgery was significantly increased over peripheral surgery cases. Others have shown that hypoxia which is not due to hypoventilation is a universal response to abdominal surgery, and lasts for at least 7 days.[43] It has also been demonstrated that increased pulmonary vascular resistance, which could set the stage for intrapulmonary shunting and hypoxia, follows both accidental trauma and the trauma of surgery.[44] This hypoxia could explain the apparent congestive heart failure on a renal basis,[37] and could account for the cardiac arrhythmias on the basis of hypoxia.[28–36] Others have shown that pulmonary complications are largely related to the site of surgery.[38,45,46]

The type of anesthesia had no significant effect on the rates of postoperative complications. Controlled ventilation, however, does appear to be important in reducing cardiac arrhythmias.[19,20] Knapp[8] also found no relationship between the type of anesthesia and the development of postoperative cerebrovascular accidents or acute myocardial infarction. Others likewise found no correlation between the type of anesthesia and cardiac complications.[7,47]

Acknowledgements

I wish to thank Miss Alice Kinne and the staff of the Medical Records Department for their invaluable assistance, and Mr. John LaCasse of Maine's Regional Medical Program for his financial and technical assistance in developing the methods used here for the recording and analysis of the numerical data.

References

1. Lochhead, R. P., Coakley, C. S., Evans, J. M.: The risk of major surgery in patients with coronary artery disease. Amer. J. Med. Sci. 227:624-627, 1954.
2. Dana, J. B., Ohler, R. L.: Influence of heart disease on surgical risk. JAMA 162:878-880, 1956.
3. Butler, S., Feeney, N., Levine, S. A.: The patient with heart disease as a surgical risk. JAMA 95:85-91, 1930.
4. Morrison, D. R.: The risk of surgery in heart disease. Surg. 23:561-570, 1948.
5. Wasserman, F., Bellet, S., Saicheck, R. P.: Postoperative myocardial infarction – report of twenty-five cases. N. Engl. J. Med. 252:967-974, 1955.
6. Thompson, G. J., Panayotis, P. K., Connolly, D. C.: Transurethral prostatic resection after myocardial infarction. JAMA 182:908-911, 1962.
7. Arkins, R., Smessaert, A. A., Hicks, R. G.: Mortality and morbidity in surgical patients with coronary artery disease. JAMA 190:485-488, 1964.
8. Knapp, R. B., Topkins, M. J., Artusio, J. F., Jr.: The cerebrovascular accident and coronary occlusion in anesthesia. JAMA 182:332-334, 1962.
9. Alexander, S.: Surgery in the cardiac patient. Surg. Clin. N. Amer. 50:567-573, 1970.
10. Christie, R. V.: Dyspnea in relation to the viscoelastic properties of the lung. Proc. Royal Soc. Med. 46:381-386, 1953.
11. Sharp, J. T., Griffith, G. T., Bunnell, I. L., et al: Ventilatory mechanics in pulmonary edema in man. J. Clin. Invest. 37: 111-117, 1958.
12. Sharp, J. T., Bunnell, I. L., Griffith, G. T., et al: The effects of therapy on pulmonary mechanics in human pulmonary edema. J. Clin. Invest. 40:665-672, 1961.
13. Chiernack, R. M., Cuddy, T. E., Armstrong, J. B.: Significance of pulmonary elastic and viscous resistance in orthopnea. Circ. 15:859-864, 1957.
14. Miller, A., Chusid, E. L., Samortin, T. G.: Acute reversible acidosis in cardiogenic pulmonary edema. JAMA 216:1315-1318, 1971.
15. Anthonisen, N. R., Smith, H. J.: Respiratory acidosis as a consequence of pulmonary edema. Ann. Intern. Med. 62: 991-999, 1965.
16. Agostini, A.: Acid-base disturbances in pulmonary edema. Arch. Intern. Med. 120:307-310, 1967.
17. Avery, W. G., Samet, P., Sackner, M. A.: The acidosis of pulmonary edema. Amer. J. Med. 48:320-324, 1970.
18. Katz, R. L., Bigger, J. T., Jr.: Cardiac arrhythmias during anesthesia and operation. Anesth. 33:193-213, 1970.
19. Reinekainen, M., Pontinen, P.: On cardiac arrhythmias during anesthesia and surgery. Acta. Med. Scan. 180: Suppl. 457, 1966.
20. Bird, C. G., Hayward, I., Howells, T. H., et al: Cardiac arrhythmias during thyroid surgery. Anesth. 24:180-189, 1969.
21. Lurie, A. A., Jones, R. E., Linde, H. W., et al: Cyclopropane anesthesia: cardiac rate and rhythm during steady levels of cyclopropane anesthesia at normal and elevated end-expiratory carbon dioxide tensions. Anesth. 19:457-472, 1958.
22. Johnstone, M.: Cyclopropane anesthesia and ventricular arrhythmias. Brit. Heart J. 12:239-244, 1950.
23. Holmdahl, M. H.: Pulmonary uptake of oxygen, acid-base metabolism, and circulation during prolonged apnea. Acta. Chir. Scand.: Suppl. 212, 1956.
24. Price, H. L., Lurie, A. A., Jones, R. E., et al: Cyclopropane anesthesia: epinephrine and norepinephrine in initiation of ventricular arrhythmias by carbon dioxide inhalation. Anesth. 19:619-630, 1958.

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Intermittent Positive Expiratory Pressure in a Case of Contused Lung

JOHN R. HEWSON, M.D.*

As pointed out by Pontoppidan et al[1] in their recent review of acute respiratory failure, the use of positive end-expiratory pressure (PEEP) is becoming commonplace in the management of patients on respirators for acute respiratory failure. Gregory et al[2] reported the use of continuous positive airway pressure with spontaneous ventilation in infants suffering from the idiopathic respiratory distress syndrome. A recent report indicates that positive expiratory pressure with spontaneous ventilation is effective in correcting the hypoxemia associated with chemically induced pulmonary edema in dogs.[3]

The present communication describes the use of intermittent positive expiratory pressure (IPEP) with spontaneous ventilation in a case of contused lung with recurrent pneumothorax. The results suggest that intermittent positive expiratory pressure may have a place in the therapy of the contused lung.

CASE REPORT

A 23-year-old male sustained fractures of the third, fourth, and fifth ribs posteriorly on the right, and a fracture of the right clavicle, in an automobile accident. A 50 percent pneumothorax initially present was cleared with an intrapleural catheter in the right thorax connected to the usual type of water seal suction. In spite of the continuous intrapleural negative pressure, a chest x-ray taken at 48 hours after the accident (Fig. 1) revealed a recurrent pneumothorax, and loculated fluid around the upper lobe region on the right. The right lung appeared more dense than the left, and along the right cardiac border was a several millimeter wide lucent area similar to the 'kinetic border sign' seen in pulmonary edema. Arterial pO_2 on room air was 34 torr at this time. The patient was given 15 minutes of 10 cm. H_2O positive expiratory pressure with spontaneous breathing through a mouthpiece- valve-tube arrangement, the latter submerged in 10 cm. of water. This raised the pO_2 to 37 torr. After 15 minutes of normal breathing the pO_2 had fallen to 35 torr, and ten more minutes of the same positive expiratory pressure raised the pO_2 to 37 torr again. The patient was then placed on a regimen of intermittent positive expiratory pressure (IPEP) of 10 cm. H_2O for 10 minutes each hour, which he administered himself. In the interim, he received 4 liters of oxygen per minute via nasal prongs. Over the ensuing thirty hours, the pneumothorax disappeared, the pleural fluid decreased, the increased density of the right hemithorax diminished, and the 'kinetic border sign' disappeared (Fig. 2). The pO_2 on room air at this time was up to 46 torr, while the pCO_2 ranged between 30 and 32 mm. Hg., and the pH between 7.49 and 7.53 during this thirty hour period. After this thirty hours, the frequency of the IPEP was reduced to every two hours, and after another twelve hours it was discontinued. One day later the chest tube was removed, at which time the density of the right chest was normal although fluid obscured the right diaphragm. The pO_2 was up to 52 torr on room air at this time. The pneumothorax did not recur. Over the next five days the pleural fluid on the right decreased to almost nothing and the

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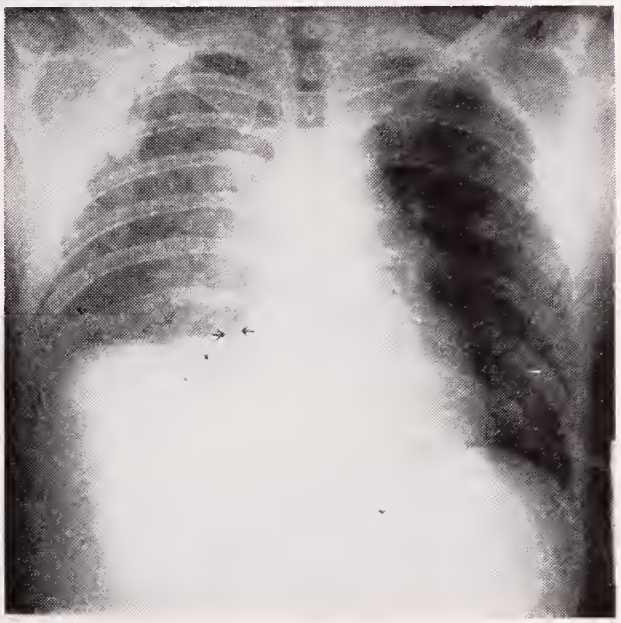

Fig. 1. A-P chest x-ray 2 days after injury showing recurrent pneumothorax (arrow at right base), increased density of the right lung, and the 'kinetic border sign' (paired arrows at right cardiac border), in spite of intrapleural catheter and suction.

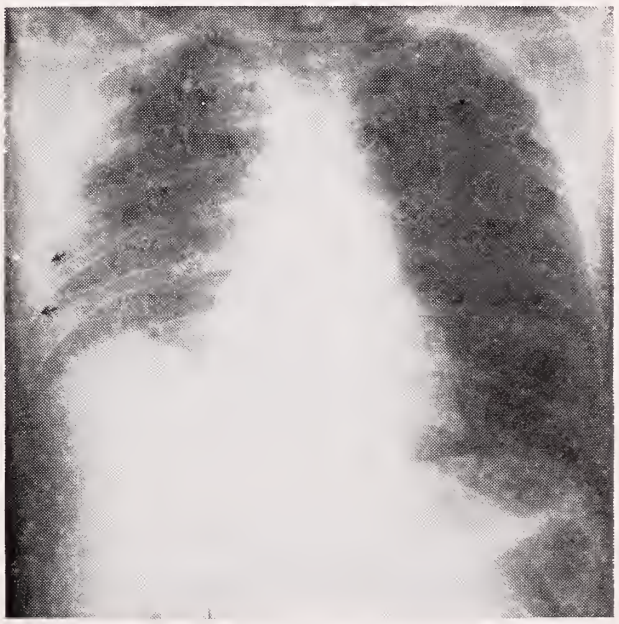

Fig. 2. A-P chest x-ray 30 hours after instituting intermittent positive expiratory pressure (IPEP), showing disappearance of both the pneumothorax and the 'kinetic border sign,' decreased density of the right lung, and remaining intrapleural fluid (arrows).

Fig. 3. P-A chest x-ray taken 8 days after instituting intermittent positive expiratory pressure (IPEP) for 42 hours, revealing only minimal remaining intrapleural fluid (arrow), and a normal lung density. (The metallic densities are related to a brace for the fracture of the right clavicle.)

right lung continued to look normal (Fig. 3). The patient was discharged eight days after starting IPEP.

REFERENCES

1. Pontoppidan, H., Geffin, B., Lowenstein, E.: Acute respiratory failure in the adult. N. Engl. J. Med. 287:799-806, 1972.
2. Gregory, G. A., Kitterman, J. A., Phibbs, R. H., et al: Treatment of the idiopathic respiratory-distress syndrome with continuous positive airway pressure. N. Engl. J. Med. 284:1333-1340, 1971.
3. Lyons, D. F., Cheney, F. W., Jr.: End-expiratory pressure in dogs with pulmonary edema breathing spontaneously. Anesthesiology 37:518-522, 1972.

CONGENITAL ANOMALIES OF THE GALLBLADDER

Continued from Page 72

uncommon condition with fifty-two previously reported cases of double gallbladder and twelve cases of previously reported cases of bilobed gallbladder. The case presentations also point out that the diagnosis is not always easy to establish, preoperatively or even at the time of surgery. The author suggests that this diagnosis might be considered in cases of recurrent common duct stones following cholecystectomy.

REFERENCES

1. Hobby, J. A. E.: Bilobed Gallbladder. Brit. J. Surg., 57: 870-872, 1970.
2. Palmisano, Donald J.: Double Gallbladder. Am. J. of Surg., 118:463-466, 1969.

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25. Black, G. W., Linde, H. W., Dripps, R. D., et al: Circulatory changes accompanying respiratory acidosis during halothane (Fluothane) anesthesia in man. Brit. J. Anaesth. 31: 238-246, 1959.
26. Sechzer, P. H., Egbert, L. D., Linde, H. W., et al: Effect of carbon dioxide inhalation on arterial pressure, electrocardiogram, plasma catecholamines, and 17-OH corticosteroids in normal man. J. Appl. Physiol. 15:454-458, 1960.
27. Camarata, S. J., Weil, M. H., Hanashiro, P. K., et al: Cardiac arrest in the critically ill. Circ. 44:688-695, 1971.
28. Downing, S. E.: Autonomic influences on cardiac function in systemic hypoxia. Proc. Int. Symp. Cardiovasc. Resp., pp 208-231, 1965.
29. Austen, W. G., Ebert, P. A., Greenfield, L. J.: Mechanism of cardiac arrest in acute hypoxia. Surg. 53:784-791, 1963.
30. Gowdy, C. W.: The autonomic nervous system in hypoxia. Proc. Int. Symp. Cardiovasc. Resp., pp 232-247, 1965.
31. Hatcher, J. D., Jennings, D. B.: Evidence for the role of humoral mechanisms in the cardiovascular responses to hypoxia and anemia. Proc. Int. Symp. Cardiovasc. Resp., pp 174-190, 1965.
32. Nahas, G. G., Mather, G. W., Wargo, J. D. M., et al: Influence of acute hypoxia on sympathectomized and adrenalectomized dogs. Amer. J. Physiol. 177:13-15, 1954.
33. Fowler, N. D., Shabetai, R., Holmes, J. C.: Adrenal medullary secretion during hypoxia, bleeding and rapid intravenous infusion. Circ. Res. 9:427-435, 1961.
34. Greenfield, L. J., Ebert, P. A.: Cardiac denervation effect in hypoxia and hypercapnea: effect of total denervation on cardiovascular responses. Arch. Surg. 87:717-721, 1963.
35. Kohler, R. L., Goldblatt, A., Braunwald, E.: The effects of acute hypoxia on the systemic venous and arterial systems and on the myocardial contractile force. J. Clin. Invest. 41: 1553-1563, 1962.
36. Downing, S. E., Mitchell, J. H., Wallace, A. G.: Cardiovascular responses to ischemia, hypoxia, and hypercapnea of the central nervous system. Amer. J. Physiol. 204:881-887, 1963.
37. Kilburn, K. H., Dowell, A. R.: Renal function in respiratory failure: effects of hypoxia, hyperoxia, and hypercapnea. Arch. Intern. Med. 127:754-762, 1971.
38. Stein, M., Cassara, E. L.: Preoperative pulmonary evaluation and therapy for surgery patients. JAMA 211:787-790, 1970.
39. Middleton, E., Jr.: The anatomical and biochemical basis of bronchial obstruction in asthma. Ann. Intern. Med. 63: 695-714, 1965.
40. Weng, T. R., Levison, H.: Pulmonary function in children with asthma at acute attack and symptom-free status. Amer. Rev. Resp. Dis. 99:719-728, 1969.
41. Weng, T. R., Langer, H. M., Featherby, E. A., et al: Arterial blood gas tensions and acid-base balance in symptomatic and asymptomatic asthma in childhood. Amer. Rev. Resp. Dis. 101:274-282, 1970.
42. Valentine, M. D.: The asthmatic patient as a surgical risk. Surg. Clin. N. Amer. 50:631-635, 1970.
43. Latimer, R. G., Dickman, M., Day, W. C., et al: Ventilatory patterns and pulmonary complications after upper abdominal surgery determined by preoperative and postoperative computerized spirometry and blood gas analysis. Amer. J. Surg. 122:622-632, 1971.
44. Kim, S. I., Shoemaker, W. C.: Development of pulmonary hemodynamic and functional changes after operative and accidental trauma. Surg. Gynecol. Obstet. 133:617-620, 1971.
45. Elwyn, H.: Postoperative pneumonia. JAMA 79:2154-2158, 1922.
46. Anscombe, A. R.: Pulmonary complications of abdominal surgery. Chicago: Year Book Medical Publishers, Inc., 1957, p 121.
47. Etsten, B., Proger, S.: Operative risk in patients with coronary heart disease. JAMA 159:845-848, 1955.

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Precautions: Do periodic serum electrolyte and BUN determinations. Do periodic hematologic studies in cirrhotics with splenomegaly. Antihypertensive effects may be enhanced in postsympathectomy patients. The following may occur: hyperuricemia and gout, reversible nitrogen retention, decreasing alkali reserve with possible metabolic acidosis, hyperglycemia and glycosuria (diabetic insulin requirements may be altered), digitalis intoxication (in hypokalemia). Use cautiously in surgical patients. Concomitant use with antihypertensive agents may result in an additive hypotensive effect.

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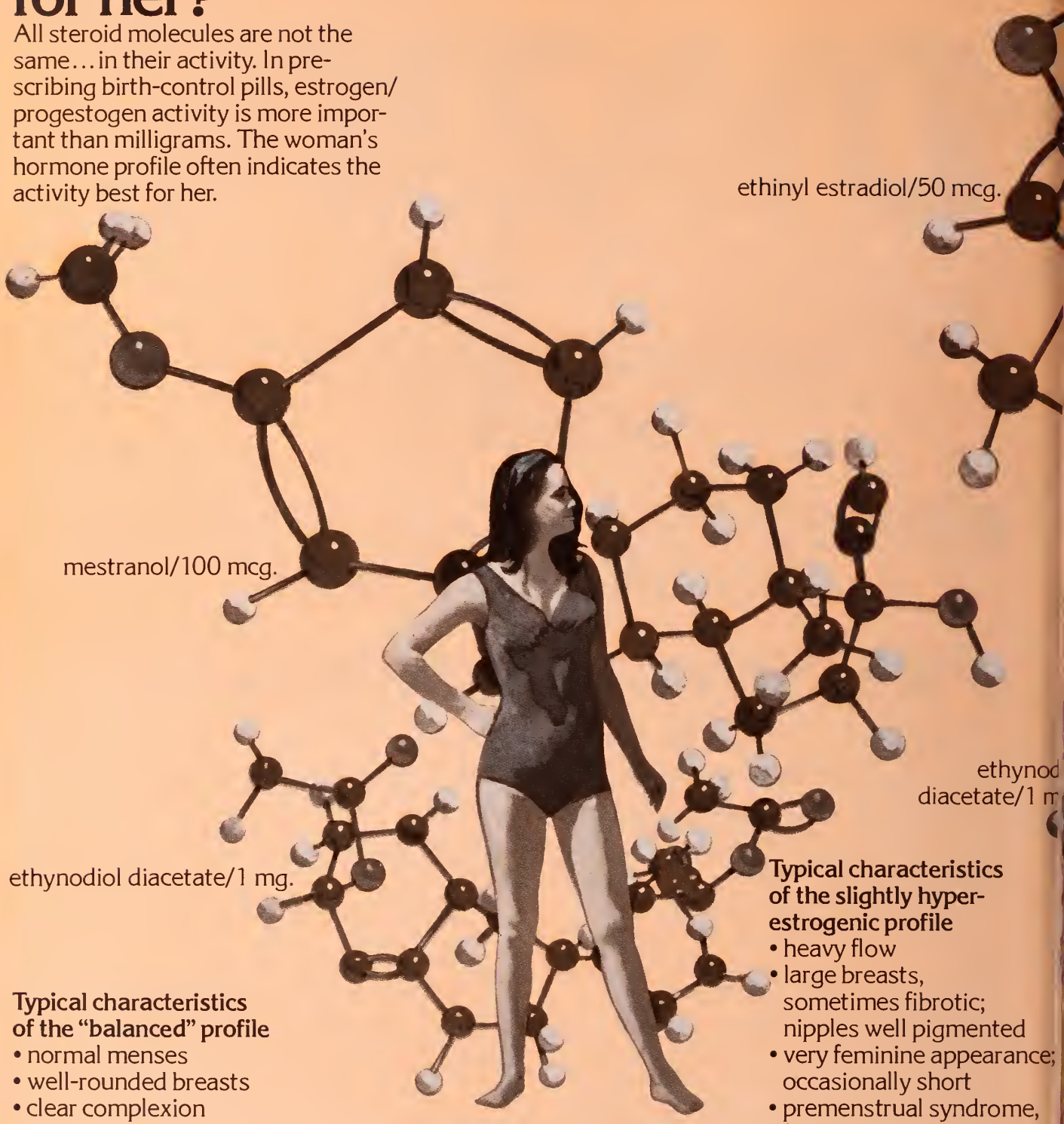

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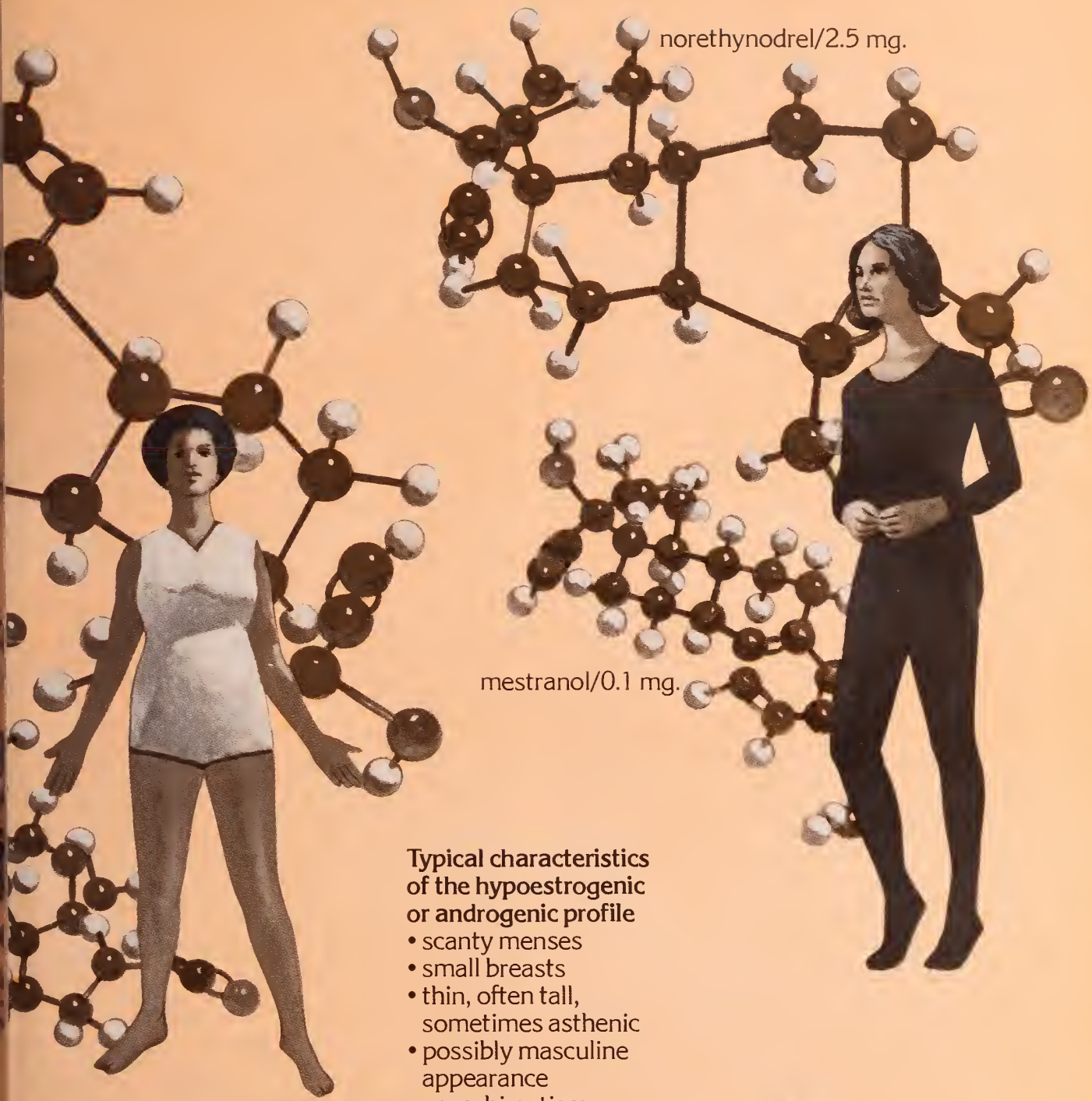

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- acne, hirsutism
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- thin vaginal lining, tendency to vaginitis and dyspareunia

This pill has a relatively weak and unique* progestogen with inherent estrogenicity. Clinically, just as in animal studies, it appears not to possess antiestrogenic and androgenic activity.

Demulen®

Available in 21- and 28-pill schedules
Each white tablet contains: ethynodiol diacetate 1 mg./ethinyl estradiol 50 mcg. Each pink tablet in Demulen-28* is a placebo containing no active ingredients.

well suited to most women when low estrogenic activity and moderate progestogen dominance are preferred

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a clear choice for women when estrogen dominance and no androgenic activity are preferred

*Of all the progestogens, norethynodrel most resembles the molecular structure of the estrogens. It has the weakest progestational activity of any progestogen in a combination pill.

Ovulen®

Each white tablet contains:
ethynodiol diacetate 1 mg./mestranol 0.1 mg.

Demulen®

Each white tablet contains:
ethynodiol diacetate 1 mg./ethinyl estradiol 50 mcg.

Each pink tablet in Ovulen-28® and Demulen-28® is a placebo, containing no active ingredients.

Actions—Ovulen and Demulen act to prevent ovulation by inhibiting the output of gonadotropins from the pituitary gland. Ovulen and Demulen depress the output of both the follicle-stimulating hormone (FSH) and the luteinizing hormone (LH).

Special note—Oral contraceptives have been marketed in the United States since 1960. Reported pregnancy rates vary from product to product. The effectiveness of the sequential products appears to be somewhat lower than that of the combination products. Both types provide almost completely effective contraception.

An increased risk of thromboembolic disease associated with the use of hormonal contraceptives has now been shown in studies conducted in both Great Britain and the United States. Other risks, such as those of elevated blood pressure, liver disease and reduced tolerance to carbohydrates, have not been quantitated with precision.

Long-term administration of both natural and synthetic estrogens in subprimate animal species in multiples of the human dose increases the frequency of some animal carcinomas. These data cannot be transposed directly to man. The possible carcinogenicity due to the estrogens can be neither affirmed nor refuted at this time. Close clinical surveillance of all women taking oral contraceptives must be continued.

Indication—Ovulen and Demulen are indicated for oral contraception.

Contraindications—Patients with thrombophlebitis, thromboembolic disorders, cerebral apoplexy or a past history of these conditions, markedly impaired liver function, known or suspected carcinoma of the breast, known or suspected estrogen-dependent neoplasia and undiagnosed abnormal genital bleeding.

Warnings—The physician should be alert to the earliest manifestations of thrombotic disorders (thrombophlebitis, cerebrovascular disorders, pulmonary embolism and retinal thrombosis). Should any of these occur or be suspected the drug should be discontinued immediately.

Retrospective studies of morbidity and mortality conducted in Great Britain and studies of morbidity in the United States have shown a statistically significant association between thrombophlebitis, pulmonary embolism, and cerebral thrombosis and embolism and the use of oral contraceptives. There have been three principal studies in Britain[1-3] leading to this conclusion, and one[4] in this country. The estimate of the relative risk of thromboembolism in the study by Vessey and Doll[3] was about sevenfold, while Sartwell and associates[4] in the United States found a relative risk of 4.4, meaning that the users are several times as likely to undergo thromboembolic disease without evident cause as nonusers. The American study also indicated that the risk did not persist after discontinuation of administration and that it was not enhanced by long-continued administration. The American study was not designed to evaluate a difference between products. However, the study suggested that there might be an increased risk of thromboembolic disease in users of sequential products. This risk cannot be quantitated, and further studies to confirm this finding are desirable.

Discontinue medication pending examination if there is sudden partial or complete loss of vision, or if there is a sudden onset of proptosis, diplopia or migraine. If examination reveals papilledema or retinal vascular lesions medication should be withdrawn.

Since the safety of Ovulen and Demulen in pregnancy has not been demonstrated, it is recommended that for any patient who has missed two consecutive periods pregnancy should be ruled out before continuing the contraceptive regimen. If the patient has not adhered to the prescribed schedule the possibility of pregnancy should be considered at the time of the first missed period.

A small fraction of the hormonal agents in oral contraceptives has been identified in the milk of mothers receiving these drugs. The long-range effect to the nursing infant cannot be determined at this time.

Precautions—The pretreatment and periodic physical examinations should include special reference to the breasts and pelvic organs, including a Papanicolaou smear since estrogens have been known to produce tumors, some of them malignant, in five species of subprimate animals. Endocrine and possibly liver function tests may be affected by treatment with Ovulen or Demulen. Therefore, if such tests are abnormal in a patient taking Ovulen or Demulen, it is recommended that they be repeated after the drug has been withdrawn for two months. Under the influence of progestogen-estrogen preparations pre-existing uterine fibromyomas may increase in size. Because these agents may cause some degree of fluid retention, conditions which might be influenced by this factor, such as epilepsy, migraine, asthma, cardiac or renal dysfunction, require careful observation. In breakthrough bleeding, and in all cases of irregular bleeding per vaginam, nonfunctional causes should be borne in mind. In undiagnosed bleeding per vaginam adequate diagnostic measures are indicated. Patients with a history of psychic depression should be carefully observed and the drug discontinued if the depression recurs to a serious degree. Any possible influence of prolonged Ovulen or Demulen therapy on pituitary, ovarian, adrenal, hepatic or uterine function awaits further study. A decrease in glucose tolerance has been observed in a significant percentage of patients on oral contraceptives. The mechanism of this decrease is obscure. For this reason, diabetic patients should be carefully observed while receiving Ovulen or Demulen therapy. The age of the patient constitutes no absolute limiting factor, although treatment with Ovulen or Demulen may mask the onset of the climacteric. The pathologist should be advised of Ovulen or Demulen therapy when relevant specimens are submitted. Susceptible women may experience an increase in blood pressure following administration of contraceptive steroids.

Adverse reactions observed in patients receiving oral contraceptives—A statistically significant association has been demonstrated between use of oral contraceptives and the following serious adverse reactions: thrombophlebitis, pulmonary embolism and cerebral thrombosis.

Although available evidence is suggestive of an association, such a relationship has been neither confirmed nor refuted for the following serious adverse reactions: neuro-ocular lesions, e.g., retinal thrombosis and optic neuritis.

The following adverse reactions are known to occur in patients receiving oral contraceptives: nausea, vomiting, gastrointestinal symptoms (such as abdominal cramps and bloating), breakthrough bleeding, spotting, change in menstrual flow, amenorrhea during and after treatment, edema, chloasma or melasma, breast changes (tenderness, enlargement and secretion), change in weight (increase or decrease), changes in cervical erosion and cervical secretions, suppression of lactation when given immediately post partum, cholestatic jaundice, migraine, rash (allergic), rise in blood pressure in susceptible individuals and mental depression.

Although the following adverse reactions have been reported in users of oral contraceptives, an association has been neither confirmed nor refuted: anovulation post treatment, premenstrual-like syndrome, changes in libido, changes in appetite, cystitis-like syndrome, headache, nervousness, dizziness, fatigue, backache, hirsutism, loss of scalp hair, erythema multiforme, erythema nodosum, hemorrhagic eruption and itching.

The following laboratory results may be altered by the use of oral contraceptives: hepatic function: increased sulfobromophthalein retention and other tests; coagulation tests: increase in prothrombin, Factors VII, VIII, IX and X; thyroid function: increase in PBI and butanol extractable protein bound iodine, and decrease in T^3 uptake values; metyrapone test and pregnanediol determination.

References: 1. Royal College of General Practitioners: Oral Contraception and Thrombo-Embolic Disease, J. Coll. Gen. Pract. *13*:267-279 (May) 1967. **2.** Inman, W. H. W., and Vessey, M. P.: Investigation of Deaths from Pulmonary, Coronary, and Cerebral Thrombosis and Embolism in Women of Child-Bearing Age, Brit. Med. J. 2:193-199 (April 27) 1968. **3.** Vessey, M. P., and Doll, R.: Investigation of Relation Between Use of Oral Contraceptives and Thromboembolic Disease. A Further Report, Brit. Med. J. 2:651-657 (June 14) 1969. **4.** Sartwell, P. E.; Masi, A. T.; Arthes, F. G.; Greene, G. R., and Smith, H. E.: Thromboembolism and Oral Contraceptives: An Epidemiologic Case-Control Study, Amer. J. Epidem. 90:365-380 (Nov.) 1969.

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Indication—Enovid-E is indicated for oral contraception.

The *Special Note, Contraindications, Warnings, Precautions* and *Adverse Reactions* listed above for Ovulen and Demulen are applicable to Enovid-E and should be observed when prescribing Enovid-E.

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Rapid Intravenous Induction Intubation Technique as Choice for Emergency Surgery

KESTUTIS KEMEZYS, M.D.

INTRODUCTION

According to Culver, the incidence of regurgitation under general anesthesia was 26 percent in 1951. This had decreased to 14 percent by 1971, probably due to newer agents and anesthetic techniques. From review of a portion of the literature, it has become obvious that emergency surgery on a full stomach can result in death of the patient due to vomiting and aspiration. All emergency surgery should be considered as surgery on a full stomach.

If it is decided to proceed with surgery under these circumstances, the following possibilities present themselves for anesthetic management.

1. Regional anesthesia.
2. Inhalation anesthesia with use of mask.
3. Inhalation anesthesia with intubation under topical while the patient is awake.
4. Intubation via tracheostomy with patient awake.
5. Inhalation anesthesia with rapid intubation technique.
6. Hypnosis.
7. Emptying of the stomach by apomorphine or tube.

It is the purpose of this paper to discuss our technique for rapid intubation under general anesthesia in the emergency situation.

TECHNIQUE OF RAPID INTRAVENOUS INDUCTION INTUBATION ANESTHESIA

The patient is oxygenated for four minutes in the waking state. This is followed by injection of a large thiopental sodium dose and immediately followed by a full paralyzing dose of succinylcholine. During this period of time, the patient is being continuously oxygenated. Forty-five seconds following succinylcholine injection, the oxygenation is stopped and the patient is intubated in a few seconds, and the endotracheal tube is inflated. The pharynx is packed with damp gauze. During this procedure if the cardiovascular status permits, the patient is tilted 40 degrees to raise the larynx 19 cms. above the gastroesophageal junction. This is sufficient to exceed the estimated 18 cms. of water intragastric pressure. At this point, the anesthesiologist should be prepared to use cricoid pressure should regurgitation develop. During oxygenation, one has to be careful not to inflate the stomach during the application of positive pressure and to use adequate doses of muscle relaxants. It is also important to test for accidental placement of the endotracheal tube into the esophagus. This is done by placing the hand below the right clavicle, and exerting pressure here while listening over the endotracheal tube.

This technique is best suited for the anesthesiologist who can intubate very rapidly. There is no room for failure in this procedure.

ALTERNATE METHODS

In general, the anesthesiologist should use the drugs and techniques with which he is most familiar. Intubation with the patient awake is a safe method. Regional or spinal anesthesia should be considered. It is unfortunate that the use of spinal and regional anesthesia meets such resistance by the layman who frequently says, "I don't want spinal or local anesthesia, I want to receive Sodium Pentothal.®" Where spinal anesthesia is indicated, vigorous educational efforts may be worthwhile to demonstrate its advantage to the patient. We must remember, however, that spinal anesthesia has its disadvantages and does not prevent aspiration in heavily sedated individuals.

CONCLUSION

Anesthesiologists should be aware of the above mentioned techniques. They should use the technique that they know best. Our data have shown a very low incidence of regurgitation and aspiration. Patient satisfaction was high with the use of rapid intravenous induction. Packing of the pharynx with slightly moist absorbable gauze blocks and absorbs any significant regurgitation. Extubation may be performed immediately following surgery or the tube may be left in place until swallowing reflexes return.

REFERENCES

1. W. K. Bannister, A. Sattilaro: Anesthesiology, March 1962, Vomiting and Aspirations During Anesthesia.
2. C. Blitt, H. Gutman, D. Cohen: Silent Regurgitations and Aspirations During General Anesthesia, Anesthesia and Analgesia Current Researches, October, 1970.
3. J. Moore and J. Fraser: The Full Stomach, Journal of the National Medical Association, January, 1971.
4. M. R. Salem: Anesthetic Management of Patient with a Full Stomach, A Critical Review, Anesthesia and Analgesia Current Researches, January 1971.
5. L. C. Jenkins and D. Lahay: Central Mechanisms of Vomiting related to catecholamine response, Anesthetic implications, Canad. Anesth. Soc., July, 71.

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Idiopathic Retroperitoneal Fibrosis

VINCENTE L. SY, M.D.

Since the initial report in 1948 by Ormond[1] of his two cases in the Journal of Urology 1948, there were more than 200 cases reported in the English Journals. Prior to the 2 cases, he reviewed cases back in 1905 by Albarran of ureteral envelopment with obstruction caused by a fibrous mass in the pelvis which was relieved by ureterolysis.

CASE REPORT

A 44-year-old white male was admitted to the Redington-Fairview General Hospital on February 2, 1971. He had been in good health until December of 1970 when he first started to notice general aches all over but especially in his back – a flu-like discomfort which did not incapacitate. Occasionally he noticed urinary frequency and urgency with the passage of mucous threads. When he came home from work he was tired and lying down for a short while made him feel better. He had a gnawing dull backache that went to his low back. In February 1971, he was admitted for study by another doctor because of a nagging backache. At this time it was more on the right flank.

Physical examination on 2 Feb. 71 revealed a healthy, well preserved male, weight was 152 lbs., normal blood pressure, pulse, respiration, and temperature. Both flanks and CVA's were slightly tender to percussion.

Laboratory Data – WBC 8000; Hgb. 94%; 47 Hct.; differential 65 polys; 32 lymph; 3 stabs; bld. Sed Rate 20.

Urinalysis not remarkable.

IVP on 2/17/71 reported that there is delayed and partial filling of the collecting system of both kidneys; there is deformity at the (Figure 1) uretero-pelvic junction.[4] This patient went home and came back on February 21, 1971. His backaches continued. Physical examination indicated nothing significant outside of the CVA's and flank tenderness to percussion.

His laboratory tests revealed – WBC 9400; differential polys 65%; lymphs 26%; monos 6%; eosin 2% and 1% basophil; urinalysis 17-22 WBC HPF; Specific gravity 1.022 – sugar negative; albumin negative. Retrograde pyelogram was done on February 22, 1971. Both #5F catheters were introduced easily into each kidney pelvis and hydronephrotic drip of clear urine was noted. Cultured urine from both kidneys revealed staph – coagulase negative – bladder urine grew E. coli. Sensitivity tests were also done. VDRL was non-reactive and Sed. Rate was 16 mm/hr. Retrograde pictures (Figure 2).

BaEnema was normal on March 1, 1971; retroperitoneal fibrosis was considered after these studies. An exploratory laparotomy was done on March 3, 1971 by an intraperitoneal approach. Both ureters were dilated in their upper third. They were bound by adhesions retroperitoneally; and these were carefully freed in their distal 2/3rd and the ureters were displaced into the peritoneal cavity by closing the peritoneal rent behind each ureter. Biopsy was taken from each side of the ureter space.

Intra-abdominal organs and pelvis were inspected and no gross pathology found. Pathological report of Dr. Richard Taylor – retroperitoneal fibrosis both right and left. Postoperatively he did well and was released from the hospital on March 14, 1971 to return as an outpatient. IVPs were subsequently followed.

COMMENTS

This disease has been reported more frequently since the original report by Ormond in the English literature. Striking symptoms of oliguria and anuria will narrow down the suspicion to the urological system. Vague back pain, leg discomfort and pelvic distress especially in female subjects will involve many other services of orthopedist, gynecologist and urologist. Even then, when the pattern of the ureters is not typical, as seen in the early stages of the disease, the diagnosis is very difficult to make. Only repeated x-ray examinations can bring it clearly to the surface. Bilateral involvement of the ureters usually indicates a retroperitoneal problem. Urograms will show typical medial deviation and stoppage of the dye. The disease is usually bilateral. Male is more common in a 3:1 ratio.

In all the cases presented by Suby, Kerr, Jr., Graham and Fraley,[2] the ureters were explored and lysed from the fibrous tissue engulfment. Several approaches were done with flank incision in the involved side; intraperitoneal routes were also used to lyse both ureters. The value of the intraperitoneal approach should be emphasized for dealing with this disease; even with apparent involvement of only one side. Sooner or later the unobstructed ureter will be involved. Harrow and Sloans[6] relieved one side and later had to relieve the other side; Hume, Erb, and Stevens[3] of Philadelphia encountered the same situation. Both ureters should be lysed and replaced laterally whether obstructed or not. Jore, John[10] has 3 cases of recurrence in the same ureter with intraperitoneal transposition but succeeded only by transfixing the ureters laterally from the original course.

The causes of this illness are all presumptive: inflammatory condition seems to top the list. Anti-serotonin medication has to be watched carefully. Lysis of the ureter seem to be the mainstay of the treatment.

REFERENCES

1. Ormond, J. K.: Bilateral Obstruction Due to Envelopment and Compression by an Inflammatory Retroperitoneal Process, Journal of Urology, Vol. 59; pp. 1072-1079, June 1948.
2. Suby, Kerr, Jr., Graham and Fraley: Retroperitoneal Fibrosis: A Missing Link in the Chain, Journal of Urology, Vol. 93; pp. 144-152, 1965.
3. Hume, Erb and Stevens: Idiopathic Retroperitoneal Fibrosis, Vol. 105; pp. 699-701, American Journal of Surgery 1963.
4. Hawk, M.D., Hazard, M.D.: Sclerosing Retroperitonitis and Sclerosing Mediastinitis, Vol. 32; pp. 321-334, October 1959.
5. Schneider, M.D.: Idiopathic Retroperitoneal Fibrosis Producting Vena Canal, Biliary Ureteral and Duodenal Obstruction, Vol. 159; pp. 316-320, American Journal of Surgery 1964.
6. Harrow, M.D. and Sloans, M.D.: Idiopathic Retroperitoneal Fibrosis, Vol. 38; pp. 148-152, JAMA, October 1962.
7. Turner, M.D. and Walker, M.D.: Idiopathic Retroperitoneal Fibrosis, Vol. 40; pp. 452-455, Journal of International College of Surgeons, November 1963.
8. Goldberg, M.D.: Idiopathic Fibrosis Demonstrating a Marked Polyuric Phase, Vol. 87; pp. 618-619, Canadian Medical Association Journal, September 1962.
9. Hache', M.D., Utz, M.D. and Woalner, M.D.: Idiopathic

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Fig. 1

Fig. 2

From the Secretary's Notebook

1972 Fall Meeting of the M.M.A. House of Delegates

The Fall Meeting of the M.M.A. House of Delegates was held at St. Joseph Hospital in Bangor, Maine on Sunday, December 10, 1972 with an attendance of 35 delegates and alternates and three guests. George W. Wood, III, M.D., President of the M.M.A., called the meeting to order and George W. Bostwick, M.D., Speaker of the House, presided.

1. **A resolution from Hancock County,** asking that the M.M.A. send annual forms to all medical practitioners in Maine, enabling them to succinctly express their views on the usefulness and cost of the activities of such governmental bodies as BNDD and quasi governmental bodies which act as fiscal intermediaries; such form to be sent to the congressmen and State legislator for the practitioner's area, and that the M.M.A. send letters explaining the intent of this action to all members of the State legislature and the Maine congressmen, was *defeated,* with only the delegate from Hancock County in favor.

2. **Report of AMA Clinical Convention, Cincinnati, 11/72** – Robert E. McAfee, M.D., alternate delegate to the AMA, summarized the many items presented at this meeting. For years, he said, many physicians have felt that some of the AMA committees and councils are redundant and costly. At this meeting, the Board of trustees reacted and 4 councils and 6 committees were eliminated. This proved, Dr. McAfee added, that persistence can change things. Since its June annual meeting, the AMA House of Delegates had 110 items and 60+ resolutions to consider – among them PSRO, H.R. 1, the Medicredit Bill, unified membership, the formation of a separate Division of Emergency Medical Services, and HMO's. The AMA supports the concept of moratorium on HMO – it should not be considered as the exclusive provider of medical care. Dr. McAfee also announced that AMA specialty journals will be free to AMA members only until June 30 – then they will be available by subscription only.*

Dr. McAfee reported that Dr. Darlington, Chairman of the M.M.A. Legislative Committee, made an excellent presentation at this meeting, on the **chiropractic situation in Maine.** He commented on the Wisconsin Study Report which outlaws chiropractic in that State. A copy of this report was sent to the President and Secretary of each County Medical Society in Maine, and to the members of the M.M.A. Executive Committee.

3. *Committee Reports:*

Care of the Disadvantaged – Dr. David Dixon recently resigned as chairman of this committee so no followup report on medical care provided by the Army at the Indian Reservations was available.

Peer Review – A copy of Dr. Chamberlin's report was sent to each member of the House of Delegates.

Legislation – Dr. Brinton T. Darlington, Chairman, was unable to attend. Dr. Stitham reported on the Physician Assistant's bill. Two years ago, the Board of Registration of Medicine caused to be introduced, a bill which included a section for P.A.'s. As a result of this P.A. section, the entire bill was defeated. During the next special session of the legislature, the bill was reintroduced, sans the P.A. section, and it passed. Dr. Leadley, without consulting the Board of Registration of Medicine, recently drew up and submitted legislation which put the licensure of P.A.'s under the Department of Health and Welfare (Dr. Fisher, Commissioner of the Department of H & W, avowed vague knowledge of this, but said he didn't want it in the Department of H & W). Issue was taken with some parts of the bill, and Dr. Stitham said that the Board of Registration proposes to reinsert the provision originally put in to give the physicians permission to do what they're already doing, until the AMA comes up with an exam to certify and register them. Thirty states have entered legislation to hold this until then, and that is what Maine wants too, Dr. Stitham added. Dr. Stitham further stated that the Board of Registration, by statute, would be the logical one to do this, and they lean toward certification, not licensure.

Government Health Activities – Dr. John Davy, a member of this committee, reported that they met for the first time in 18 months. They felt their duties overlap in the legislative area and they have come to the realization that no one has full knowledge of fiscal affairs. Dr. Wood said that RMP has a list of government programs; also, the State has individuals responsible for knowing this list. We are waiting for a compilation of such programs and hope to pass it along to the G.H.A. committee. Dr. Wood pointed out that the new M.M.A. bylaws were set up to form common committees under one Council for less duplication (Legislative Comm. and Govt. Health Act. Comm. are under the same Council). Also, Dr. Wood added, that as an Association, we do not have the funds to put to this one thing (study of government programs).

Dr. Davy went on to say that the Government

*The AMA has recently announced they *will* continue the specialty journals as a benefit of membership.

Health Activities Committee fails because of lack of staff and perhaps the dues ought to be raised to adequately finance staff (they discussed hiring a full-time Assistant Executive Director of the M.M.A. and a secretary).

4. The Executive Committee, in October of 1972, appointed a **Committee to Investigate Having a Physician on Duty at the State House** and this committee was due to report at this meeting. Just this week, Dr. Hanley reported, the Chairman of this Committee resigned – hence, no report. This morning the Executive Committee discussed this, and voted to assign physicians to certain hours to be at the State House – but not to treat.

Many of the physicians argued that they didn't have the time to take from their practice, and questioned whether it would be effective to have a different physician there each day. Dr. Robert Andrews proposed that we pay physicians $100 a day to go to Augusta. Dr. Alfred Swett added that these are all valid approaches, but maybe we can do all three – be there, contribute through increased dues and have personal contact with the legislators. The suggestion was also made that once a year each county society invite legislators to a meeting.

Dr. Lightbody made a motion that the Executive Committee be empowered to delegate physicians, at their request, to go to legislative hearings. This was seconded and voted (with one opposed).

Dr. Davy proposed this idea for discussion – raise the dues to hire additional personnel – "$100 isn't buying anything and taking a day off is a hardship."

First, we must decide what services specifically are needed, Dr. Wood suggested, and added we should get involved ourselves and not hire the work done. Also, as a temporary measure, we should get volunteers to go to the Legislature – this may be a beginning until we can get a full-time staff.

Dr. Stitham suggested that the delegates ask their county societies if they wished to raise the State dues – up to double. A straw vote was taken to indicate who would favor an increase in dues – up to double – for hiring staff assistance. All but three voting were *in favor*.

Regarding a **lobbyist**, Dr. Hanley said that he feels it is better to see what bills are going to be in the legislature and then hire a lobbyist for each specific job. Dr. Fichtner suggested that if we did get a full-time assistant executive director, he could spend three months a year as our lobbyist.

Dr. Andrews proposed that the Executive Committee make specific recommendations as to a dues increase amount, duties of additional personnel, etc. Dr. Wood suggested that we ask each committee chairman to tell us what they think they need for support and financial assistance. The Executive Committee will discuss this at its February 1973 meeting and report back at the Interim Meeting of the House of Delegates. This was approved by the delegates.

5. Other business –

a) Dr. Fichtner reported that there is an open **seat on the Governor's Council** and he is running for it – he would appreciate the physicians' support.

b) Delegates from Cumberland County asked if **summaries of the Executive Committee meetings** could be sent to all county societies, as their District member on the E.C. is not always present at county meetings to report. This was approved and it will be done, beginning with today's meeting.

c) Dr. Wood reported that Dr. Chamberlin has been asked to devote more time to **peer review** and he will be reimbursed by the EMCRO grant. Additional staff will come from Maine's Regional Medical Program.

Re **H.R. 1**, the existing EMCRO program is being looked on as a source of information on how to do professional service review. If we choose, Dr. Wood reported, we have the opportunity of using EMCRO to develop an organ to become the PSRO for Maine. The Executive Committee has given this its vote of confidence.

Dr. Wood recently visited Michigan State with Drs. Oliver Cope and Stanley Freeman and three Osteopaths from Maine. There, 2 deans (M.D. and D.O.), are working together in the same environment. The DO's in Maine recognize that we can't afford two medical schools, Dr. Wood added. New England is developing a School of Osteopathy and Dr. Roswell Bates (Ex. Dir. of the Maine Osteopathic Association) is one of the consultants. Osteopathy in Maine may be doing for the N. E. School of Osteopathy what the MMC does for Tufts, Dr. Wood said.

PATRICIA A. BERGERON
Secretary-Treasurer, M.M.A.

IDIOPATHIC RETROPERITONEAL FIBROSIS — *Continued from Page* 80

Fibrosis Retroperitonitis, Vol. 115; pp. 737-744, Surgery, Gynecology and OB, December 1962.
10. John S. Jore, M.D., FRCS: Idiopathic Retroperitoneal Fibrosis, Vol. 39; pp. 431-443, British Journal of Urology, 1967.
11. P. J. Stueber, Jr.: Primary Retroperitoneal Inflammatory Process with Ureteral Obstruction, Vol. 82; pp. 41-50, Journal of Urology, 1959.
12. Arjan D. Amar: Ureteropyelostomy for Relief of Single Ureteral Obstruction Due to Retroperitoneal Fibrosis in a Patient With Ureteral Obstruction, Vol. 103; pp. 296-297, Journal of Urology, 1970.
13. Brann, Staubitz, Oberkircher and Nilsen: A Review of Retroperitoneal Fibrosis, Vol. 92; pp. 323-330, Journal of Urology, 1964.
14. Charnock, Riddell and Lombardo, Jr.: Retroperitoneal Fibrosis Producing Ureteral Obstruction, Vol. 85; pp. 251-257, Journal of Urology, 1961.
15. J. K. Ormond: Idiopathic Retroperitoneal Fibrosis, The Ureter, Edited by Harry Bergman, M.D., Copyright 1967, pp. 293-315.

Milford Ave., Bingham, Maine 04920.

THE HEALTH COMPONENTS OF H.R. 1

H.R. 1, the House of Representatives' bill number 1 amending the Social Security Act, has changed the coverage available under both Medicare and Medicaid. There were new people made eligible by the bill, such as the disabled who will be covered by Medicare effective July 1973. New benefits were also made available under the provisions of the bill, including speech pathology and chiropractic services.

New People Eligible

For Medicare Level of Benefits:

Automatic coverage for 1.7 million disabled (all disabled workers at any age, disabled widows and disabled widowers between the ages of 50 and 65, beneficiaries age 18 and older who receive benefits because of disability prior to age 22, the blind, plus disabled qualified Railroad Retirement annuitants), effective July 1973.

Optional coverage, by direct enrollment @ $33 for Part A plus $5.80 for Part B, for persons 65 and over who previously were ineligible for Medicare, effective July 1973.

For Medicaid Level of Benefits:

Eligibles under age 21 may now be covered for bed care in mental institutions, in specific situations.

Other:

10,000+ "fully or currently insured workers under Social Security and their dependents with chronic renal disease" would be deemed disabled and thereby be eligible for renal dialysis or kidney transplant coverage three months after such a course of dialysis begins, effective July 1973.

New Medicare Benefits

Speech pathology in participating clinics with physical therapy, effective January 1973.

Chiropractic, but only for manual manipulation of spine, effective July 1973.

Optometric services, but only for fitting prosthetic lenses.

Cost of supplies directly related to the care of a colostomy.

Some greater flexibility in defining covered services in a skilled nursing facility.

Renal dialysis and kidney transplants as described above.

Eliminates the need for the recipient to pay 20% of the cost of home-health services under Part B ($60 deductible remains).

Physical therapy services provided in therapist's office or in a hospital or ECF out-patient department ($100 limit on office/home PT), effective July 1973.

Automatic enrollment in Part B when eligible for Part A, effective July 1973.

Through experimentation, pay for medical equipment vs. rental.

Permit payments "not to exceed 100% of present Part A & B per capita costs in a given geographic area" to an HMO . . . a prepaid group practice, a foundation for medical care or a neighborhood health center meeting broad HMO guidelines effective July 1973.

Services in a foreign hospital if it is closer to the recipient's residence or more accessible than the nearest suitable U.S. hospital.

New Medicaid Benefits

"Intermediate care" in a mental institution for those 65 and over if they could also have been covered there for hospital or skilled nursing care.

Chiropractic, but only for treatment of spine by manual manipulation, effective July 1973.

Services of an optometrist in states which had been providing optometric services by an M.D.

Either family planning services . . . or Aid for Dependent Children funds reduced by 1% annually, effective July 1973.

Either child health screening services and treatment . . . or AFDC funds reduced by another 1% annually, effective July 1974.

Eligibility cannot be lost (because of cash benefit increase) until October 1973, a year's grace.

New Controls and Belt-Tightening

Precludes Medicare/Medicaid payments for capital expenditures for health facilities and services not approved by an areawide health planning agency.

Either effective utilization review of institutional services under Medicaid . . . or a one-third reduction in federal matching funds, effective July 1973.

Part B deductible goes from $50 to $60.

Minimum lengths of stay for post-hospital patients will be established *according to diagnosis* for ECF and home health purposes.

Establishes PSRO's (Professional Standards Review Organizations), effective January 1974, in local areas to review Medicare/Medicaid services for medical necessity (not reasonable charge). Must be physician-sponsored until January 1, 1976; after that date, other groups eligible.

To help pay for higher Social Security cash and health benefits, the tax rate goes from the current 5.2% to 5.85% in 1973, then 6.05% in 1978, then to 7.3% in 2011. Tax base also is raised, to produce a maximum personal tax of $631.80 annually.

Categorically indigent under Medicaid will be required to pay a monthly premium, based on their income; and states have the option to impose deductibles and co-insurance.

Maine Heart Association Notes

20 Winter Street, Augusta, Maine

Computers and Cardiovascular Medicine

James W. Covell, M.D.*

Introduction

Although the potential of computing techniques in cardiovascular medicine has been recognized for some time, practical applications have become available only recently. Advances in technology coupled with rapid growth of a pool of individuals with special skills in biomedical computing have helped to propel computers to the forefront in cardiovascular diagnosis, patient care, and research at a cost that is no longer prohibitive.

There are two fundamental types of computers — analog and digital. An analog computer consists of linked networks of electronic components capable of analyzing and characterizing signals derived directly from physiological recordings. Thus, analog computers are used to analyze and filter the electrocardiogram; recognize and record the frequency of ventricular premature contractions; calculate and record cardiac output, heart rate, systolic, diastolic, and mean arterial pressures. These devices have been effective in intensive care and coronary care units. However, their use is limited since they possess no capacity for memory and are thus unable to correlate a patient's current physiological status with other variables such as his past condition, or previous history or physical findings. Their effectiveness can be enhanced by combination with the second type of computer — the digital computer.

Digital computers are complex, high speed, costly devices. They perform a wide variety of independent operations (additions, subtractions, multiplications, etc.) in microseconds; but they perform each operation one step at a time. Thus, they require a program that essentially "tells" the computer what step to take next and where, in the circuitry, to store resultant information. Accordingly, digital computing requires first the electronic hardware — a computer, and second, the software, i.e., programs comprising instructions to the computer concerning a particular task. Both are equally essential to computer utilization.

Digital computation has been applied successfully to electrocardiographic diagnosis, patient monitoring, and the storage and retrieval of information derived from the patient's history and physical examination. Fortunately, existing hardware is now adequate for most tasks in these areas. Furthermore, numerous programs have been developed and tested, and are now commercially available.

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Specific Applications

Electrocardiographic Diagnosis. During the past several years, programs have been developed for electrocardiographic interpretation utilizing signals obtained either from standard twelve lead electrocardiographs or from vector cardiograph equipment. Naturally, some variation exists between a given physician's reading of an electrocardiogram and the computer's diagnostic analysis of the tracing. However, it is now generally accepted that computer analysis is effective in correctly classifying normal records and that good agreement between a human expert and the computer can be anticipated for most abnormal records as well. It is now possible to obtain electrocardiographic analysis by computer via ordinary telephone lines within minutes utilizing commercial services available in most areas. In some cases, computer recognition of abnormal tracings can be directly linked in order to initiate automatically additional expert consultation within large center. Naturally, the computer cannot substitute for physician judgements based on the patient's previous electrocardiograms, history, or physical examination. In the assessment of these important diagnostic factors, as well as in the assessment of small variations in the electrocardiogram wave form, the computer cannot replace but can only supplement the skilled judgement of the physician.

Patient Monitoring. Perhaps the most rewarding and eventually most important application of computer electrocardiographic analysis will be in the area of intensive care monitoring. It is well recognized that prompt treatment of bradycardia and/or ectopic ventricular beats can lead to dramatic reductions in the mortality rate from acute myocardial infarction. Since programs are now available for on-line interpretation of arrhythmias and for on-line analysis of wave form, it is now possible to utilize computer diagnosis in the intensive care setting for on-line patient monitoring. Computer analysis of signals obtained with appropriate transducers can be used to monitor constantly not only the electrocardiogram, but also respiratory function, blood pressure, and other physiological functions. Programs have been devised which enable the computer to alert nurses or attending physicians whenever specified arrhythmias or alterations in hemodynamic or respiratory status occur. Computer systems for this purpose currently commercially available cost less than $10/day/bed and can significantly supplement conventional monitoring techniques.

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AMERICAN CANCER SOCIETY-MAINE DIVISION, INC.
Brunswick, Maine

The Postmastectomy Lymphedematous Arm

GUY F. ROBBINS, M.D.* and WILLIAM M. MARKEL, M.D.**

INTRODUCTION

Postmastectomy lymphedema causes serious problems in less than ten percent of patients. Yet, when it occurs, lymphedema is a distressing and sometimes disabling burden for the patient and a challenge to the physician. Controversy continues regarding etiology and treatment programs vary. However, it is agreed that the prevention of the postmastectomy edematous arm is far more valuable than any valiant surgical or medical procedure designed to remedy the condition once it has occurred.

ETIOLOGY

An often confusing array of etiologic factors have been advanced to explain the development of lymphedema. Prime factors appear to be: (1) unusual trauma of the axillary vessels during surgery; (2) postoperative infection of the axillary area; and (3) fibrosis following radiation therapy. Postoperative infection, signalled by localized induration and occurring even years later, as the result of burns, cuts, insect bites, severe bruises of the hand, arm and forearm, hypodermic or intravenous injection or vaccination may either initiate the process or increase the severity of pre-existing lymphedema.[1]

Regardless of the specific etiology, the mechanisms producing lymphedema are essentially the same: the lymph flow from the extremity is blocked causing an increase in hydrostatic pressure. This increase in pressure leads to dilated lymph vessels and incompetent lymph valves. At the same time, there is an increase in the protein content of lymph and proliferation of fibroblasts in the tissues. And, since a lymphedematous extremity is an excellent culture medium for bacteria, resultant infection may lead to thrombosis of the lymph vessels and further blocking of lymph flow, increased lymph stasis and progressive fibrosis of the connective tissue.

PREVENTION

Preventive measures begin at the time of operation. Careful handling of tissues during the surgical procedure and the development of an incision that will not close directly over the axillary bundle are advised. Also, care should be taken not to disturb the clavicular portion of the pectoralis major muscle since it will provide a shelf-like roof for protection of the proximal portion of the axillary vein. To prevent injury to existing structures, vein thrombosis, infection or the formation of excessive cicatrix, it is important that the physician: (1) use a sharp dissection thus avoiding the necessity for mass clamping or mass ligature; (2) handle the axillary vessels gently and carefully ligate the tributaries of the axillary vein; (3) use fine suture material. Primary skin grafting may help avoid incisional scars over the axillary vessels. Specific operative procedures designed to avoid postmastectomy edema have been described by various physicians.[2]

Another important aid in the prevention of lymphedema is the maintenance of gravity drainage following surgery through elevation and partial abduction of the extremity. The patient's fingers and hand are raised higher than the forearm and the forearm higher than the pillow. Since this position cannot be maintained for the optimum period of six weeks, elastic compression bandaging from finger tips to shoulder is highly recommended for six weeks following surgery. Active motion and graduated exercises beginning on the first postoperative day are also helpful.[3] However, such exercises are not indicated when skin grafts have been used or when the patient has been subjected to a radical mastectomy and chest wall resection.

It is essential that the postmastectomy patient be instructed on the various measures to prevent trauma which may lead to impaired lymphatic and vascular circulation. The following written list of "dos and do nots" should be given to patients upon discharge:

1. Push cuticles back – do not cut – avoid possible infection.
2. Wear canvas gloves when gardening; wear rubber gloves when cleaning pots and pans with steel wool.
3. Wear padded gloves when reaching into oven. avoid burns.
4. Keep watch band and jewelry loose on operative arm.
5. Keep dress sleeves loose. Avoid pressure and swelling.
6. Use unaffected arm to carry heavy purse and packages.
7. Use unaffected arm for blood pressure readings, injections, vaccination, etc.
8. Wear thimble when sewing.
9. Wash the smallest break in the skin on operative side, immediately with soap and water, and cover with a band-aid.
10. Use electric razor for shaving and avoid nicks and scrapes.
11. Do not get sunburned; get tanned gradually.
12. Contact doctor if arm on operative side feels hot, is reddened or swollen.

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**Vice President, Service and Rehabilitation, American Cancer Society.

13. Hold cigarette in the unaffected hand – avoid possible burns.
14. Keep arm elevated when sitting; do not let arm hang down by side.

Frequent follow-up is necessary to keep the patient mindful of the need for preventive action. The use of a fitted elastic sleeve and/or one of the available units which apply controlled pressure is practiced by many clinicians and although these procedures can be useful, in-depth scientific evaluation of these products is not available.

Treatment

Despite preventive measures, approximately ten percent of postmastectomy patients develop lymphedema. Present-day chemotherapy has been helpful when infection is present; surgical procedures reported in the literature designed to re-establish lymph flow generally result only in a modicum of success. However, there is recent, encouraging data on the control of postmastectomy lymphedema by the transposition of the partially detached omentum beneath the superficial tissues of the chest wall to the axilla and upper arm.[4]

Nevertheless, it appears that for the present we must consider some kind of mechanical means to overcome the obstruction of fluid flow. Elastic sleeves are frequently helpful although they may be uncomfortable for the patient and they are sometimes cosmetically unacceptable. A pressure pump may be helpful in special instances under medical direction. However, if there are signs of lymphangitis (redness and streaking of the arm, with or without accompanying fever) manual manipulation of the arm is contraindicated.

Because of the prevalence of infection, some physicians give antibiotics prophylactically to all patients with postmastectomy lymphedema.[5] Antibiotics are advised in the acute phase of lymphangitis, for long-term treatment during the chronic phase, if there is any necrosis of the wound margin or fluid beneath the wound flap.[6] A low salt diet may help, at times, and the administration of dehydrating agents has been of limited assistance.

Summary

Postmastectomy lymphedema is a distressing and sometimes disabling complication which causes serious problems in less than ten percent of patients. All available preventive measures should be utilized since medical and surgical procedures are not very effective once the entity has occurred. The use of elastic sleeves and pressure pumps may be beneficial. Antibiotics are indicated in the acute phase of lymphangitis and for long-term treatment during the chronic phase, if there is any necrosis of the wound margin or fluid beneath the wound flap. A low salt diet and the use of diuretics may also be of assistance.

References

1. Britton, Richard C., M.D. and Nelson, Paul A., M.D.: Causes and Treatment of Postmastectomy Lymphedema of the Arm. Journal of the American Medical Association, 4/14/62, Vol. 180. Pp. 95-102.
2. Pack, G. T.: Tumors of the Breast, Chest, and Esophagus. 2nd ed. v. 4, 1960. New York, Harper & Brothers. Pp. 149-153.
3. Patterson, Mary G.: Special Nursing Problems of the Patient with Cancer: Pack, G. T. and Ariel, I. M. (eds): Treatment of Cancer and Allied Diseases, Vol. I. New York: Paul B. Hoeber, Inc., 1958. Pp. 563-570.
4. Goldsmith, Harry S.: The Treatment of Postsurgical Lymphedema. Surgical Clinics of North America, Vol. 49, No. 2, April, 1969.
5. Nelson, Paul A., M.D.: Recent Advances in Treatment of Lymphedema of the Extremities. Geriatrics 21: 162-173, 1966.
6. Dietz, J. Herbert, Jr., M.D., F.A.C.S.: Rehabilitation of the Cancer Patient, Medical Clinics of North America, Vol. 53, No. 3, May 1969.

COMPUTERS AND CARDIOVASCULAR MEDICINE – *Continued from Page* 86

Discreet Data Analysis. Another area in which computers now contribute effectively to cardiovascular medicine is that of maintaining medical records. Unfortunately, the extent of utilization of this important application has been disappointing. Theoretically, information entered in medical records can be classified and stored relatively easily with the aid of modern computing technology. Recent emphasis on the development of techniques necessary for direct computer-physician interaction and on utilization of problem oriented medical records may help to facilitate more widespread applications in this area. Computer-physician interaction can be utilized as follows: the computer is programmed to flash a question on an oscilloscope screen. The physician responds in a yes or no fashion or by typing a response in ordinary English. The computer's questions continue, usually according to a "branching" logic pattern, similar to that employed by a physician obtaining a history. Thus, the computer might inquire whether chest pain had been present. An affirmative response would lead to additional questions concerning details including its radiation, intensity, duration and frequency. Depending on the response, the computer would ask additional questions to obtain further relevant data. By this means, data can be obtained for storage and retrieval in a logical format. This approach has been applied to storage and retrieval of information in nurses notes, intake-output records, medication records, and physicians history and physical examination reports.

Future Applications

Computing techniques will undoubtedly play an expanding role in cardiovascular medicine. Widespread utilization will probably occur first in the form of on-line monitoring in coronary care units and subsequently in electrocardiogram interpretation and large scale screening. Economic application of computing techniques to facilitate information storage and retrieval will ultimately be of considerable importance in effective maintenance and utilization of medical records for patient care as well as for investigation in cardiovascular medicine.

DEAN H. FISHER, M.D.
COMMISSIONER

State of Maine

Department of Health and Welfare

Videofluoroscopy as an Aid in the Management of Velopharyngeal Insufficiency

CLEFT PALATE EVALUATION TEAM, DIVISION OF CHILD HEALTH
ROBERTA HANSEN, M.A., SPEECH AND HEARING CONSULTANT, COORDINATOR

Summary: Normal speech and voice is the goal of management in the child with velopharyngeal insufficiency (VPI). Until three years ago, examinations of oral mechanisms by physicians and of speech and voice by speech pathologists were used to determine the plan of care. Then, at the request of the Cleft Palate Team, Division of Child Health, State Department of Health and Welfare, fluoroscopic techniques for an objective evaluation of the velopharyngeal port were developed by the Department of Radiology, Maine Medical Center. The Cleft Palate Team supports the use of videofluoroscopy as an aid in the diagnosis and management of the patient with VPI.

Videofluoroscopic service is available directly through Irving L. Selvage, M.D., Department of Radiology, Maine Medical Center, Portland, or by individual referral from the Division of Child Health, State House, Augusta, Marguerite Dunham, M.D., Director.

Services for children with congenital clefts of palate and/or lip have been available through the Division of Child Health (formerly called Crippled Childrens Service), State Department of Health and Welfare, since 1936. The Cleft Palate Team,* meeting four times a year at the Maine Medical Center in Portland, was founded in 1954 to coordinate care for children with clefts who represented problems in management. A team approach, with a staff consisting of a pediatrician, a plastic surgeon, an otolaryngologist, general dentist, orthodontist, speech pathologist, social worker, public health nurse and nutritionist, remains operational, with the addition of a radiologist in 1970, and serves about twenty children under the age of eighteen each year.

In Maine, the birth rate has approached 18,000 for the last three years. Using the figure of one birth in seven hundred with some form of oral cleft, approximately twenty-five of these children are born here yearly. The Division of Child Health receives about twenty referrals a year, as well as a few referrals of children with VPI due to other causes (sub-mucous clefts, congenital short palate – sometimes unrecognized until post-tonsillectomy, neuromuscular dysfunction, etc.). This leaves about 20% of the children with known oral clefts and an unknown number of children – perhaps several hundred – with VPI due to other causes, under private care.

In the average patient, the valving action of the soft palate (velum) against the posterior wall above the plane of the atlas produces the resonance and pressure needed for normal speech. In the presence of a congenital cleft of the soft palate or palatopharyngeal incompetence, the valving process has been disrupted. Defective voice and speech result. There is nasality, an inability to produce certain speech sounds (especially 's', 'sh', 'k' and 'g') and reduced intelligibility in conversational speech. The goal of treatment is normal voice and speech. The choice of treatment depends on an analysis of how the velopharyngeal mechanism operates for each patient. Videofluoroscopic techniques described later in this paper permit sophistication in choice of management based on the ability to see relationships within the velopharyngeal port.

Choices in treatment include: (1) speech therapy, (2) push-back and/or pharyngeal flap procedures, (3) pharyngoplasty, (4) combination of pharyngeal flaps or pharyngoplasty with speech therapy, or (5) prosthesis** alone, combined with speech therapy or in preparation for surgery.

If the visualized velopharyngeal mechanism is adequate, and VPI speech is manifest, habit or psychological

*Current Cleft Palate Team: Alice Whittier, M.D., Pediatrician; Burton Olmsted, M.D., Plastic Surgeon; Marvin Adams, M.D., Otolaryngologist; Robert Hutchinson, D.D.S., General Dentistry; C. Leighton Philbrook, Orthodontist; Irving L. Selvage, M.D., Radiology; Amelia Sanders, Medical Social Worker; Dorothy Woodcock, Nutrition Consultant; Margery Groves, Public Health Nursing; Roberta Hansen, Speech and Hearing Consultant, Coordinator.

**Not currently available in Maine.

factors may be operating, and speech therapy – not surgery – is probably indicated. If we can see that velum is short and there is good lateral pharyngeal wall action, pharyngeal flap procedure is probably indicated. If the velum is short, but good lateral pharyngeal wall ability is missing, pharyngeal flap is contraindicated and pharyngoplasty or prosthesis may be the treatment of choice. Different combinations may be used, with the radiologic information clarifying the cause and effect.

It would be simplistic to maintain only these choices in management. They do indicate that determination of treatment is dependent upon the extent of information available.

The Cleft Palate Team used the professional but subjective techniques of examination of oral mechanisms by a plastic surgeon, and speech and voice analysis by a speech pathologist as the basis for determining a plan of treatment. In 1969, the Department of Radiology at the Maine Medical Center was asked to develop radiologic techniques to add dimension to the evaluation of patients scheduled for the Cleft Palate Evaluation Clinic or with a question of VPI. Initial technique involved a 16mm Cine at 30 to 50 frames per second and a Picker 9 inch image amplifier. A coating of Dionsil provided contrast, and the patient was asked to repeat a battery of phrases, sounds, and counting in lateral view. Films were of fair quality only, but provided more realistic anatomic evaluation than previously possible. Elevation and length of the velum were well defined, but action of the lateral pharyngeal wall – the predictor of success with the pharyngeal flap procedure – could not be visualized adequately. Lack of synchronized voice, and the age of the patients referred† were problems. Forty-seven cine studies were made between June 1969 and December 1970, all but four referred by the Division of Child Health.

In 1972, the technique was changed to approximate the fluoroscopic examination used by Dr. Skolnick‡ at the Upstate Medical College in Syracuse, New York, with the exception of the head immobilization devices. Implementation was available at the Maine Medical Center and the technique appeared to offer more information. Essentially, picture and voice recordings of the velopharyngeal port are recorded sequentially in lateral and base projections. A Picker 6 inch image intensifier with 1.0 mm fluoroscopic focal spot, a Plumbicon TV system, and Sony 2″ video recorder is used, with a barium paste inserted by polyethylene tube to provide contrast. The patient repeats the battery of sentences, words, sounds, and swallows. Thirty-three examinations have been made since January of 1972 with good film quality. Most patients over four years of age have tolerated the procedure well. Cooperation has been maximized by positioning the patient to see himself in the video screen – *Instant TV Star!*

Videofluoroscopic examination and interpretation in conjunction with the professional judgment of the speech pathologist reduces the subjective aspect of the management of the patient with VPI, and encourages treatment at the appropriate developmental stage. The Cleft Palate Team has found this to be a useful tool and would encourage physicians with patients in whom VPI is suspect, to consider this examination as part of their armamentarium in diagnosis and treatment.

Service is available directly through the Department of Radiology, Maine Medical Center, Portland, Maine, or may be available through the Division of Child Health, Department of Health and Welfare, State House, Augusta.

†Referral of children previous to birthday four was not effective.
‡Skolnick, M. L.: Videofluoroscopic examination of the Velopharyngeal Port During Phonation in Lateral and Base Projections – a New Technique for Studying the Mechanics of Closure, Cleft Palate. J. 7:806.

REFERENCES

1. Kelsey, C. A., Ewanowski, S. J. J., et al: Lateral Pharyngeal-Wall Motion as a Predictor of Surgical Success in Velopharyngeal Insufficiency. N.E.J. Med. 287:64-68.
2. Skolnick, M. L.: Videofluoroscopic examination of the Velopharyngeal Port During Phonation in Lateral and Base Projections – a New Technique for Studying the Mechanics of Closure, Cleft Palate.
3. Blakely, R. W.: The Rationale for Temporary Speech Prosthesis in Palatal Insufficiency. B. J. Discord Commun 4: 134-139.
4. Weber, J., Jr., Chase, R. A., et al: The Restrictive Pharyngeal Flap B. J. Plas. Surg. 23:347-351.
5. Osborne, G. S., Pruzansky, S., Koepp-Baker, H.: Upper Cervical Spine Anomalies and Osseus Nasopharyngeal Depth. J. Sp. Hear. Res. 14:14-21.

Dates For Your 1973 Calendar . . .

M.M.A. Annual Session — June 16-19

AMA Annual Convention — June 23-28

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Other Meaning of Symptoms*

Alan M. Elkins, M.D., F.A.P.A.**

Introduction

Every physician in practice has had the experience of seeing significant numbers of patients, without objective findings of organic disease. The percentage has been estimated at anywhere between 50 and 75 percent. In family practice, it is especially common to find patients who come to the physician – even with concrete physical symptoms – in whom no organic syndrome can be diagnosed. The doctor himself is aware of being frustrated under these circumstances and is often able to offer little if any help to the patient. Patients will, however, continue to present themselves to physicians under these circumstances and to anticipate some kind of help from him.

There are two obvious groups of patients who present with symptoms but are without organic disease: (1) those who have clear cut somatic symptoms, such as pain or gastrointestinal disturbance but in whom the psychiatric illness is paramount and (2) those patients who have no organic symptoms but nevertheless try to utilize the doctor-patient relationship for other reasons.

The Psychiatric Consultation Service at the Maine Medical Center sees patients from all specialties, who present with complaints ranging from pain to anxiety and depression to hallucinatory experiences and with the full range of the psychophysiologic symptoms. During a four-week period in the Fall of 1972, 33 percent of the patients were seen by the psychiatric consultant because of depression and/or anxiety. Another 16 percent presented with pain or other somatic symptomatology. The remaining 51 percent of patients seen in consultation had problems relating to drug abuse, alcoholic abuse, were suffering from organic psychoses, or had legal problems.

An attempt will be made in this paper to describe some of the relationships between symptoms and the "other meanings" that the symptoms have. Some suggestions for the approach to these patients will be made as well as some notions about management.

Pain as a Symptom of Psychiatric Illness

According to Rangell, various experimental and clinical methods are used to record the response to pain and show that it is influenced by attitude, suggestion, and emotional stimuli. He goes on to state that pain depends not only on the neural pathways mediated but also on the emotional status of the individual at the time. It has been demonstrated, for example, that pain threshold can be raised by 40 percent by hypnosis.

All of us have seen the results of using placebos judiciously and there are those who feel that the Chinese practice of acupuncture may be influenced significantly by suggestion. The response of a given individual to pain depends on past experience with pain as well as his conscious and unconscious associations to pain. A fractured extremity in an adult may be seemingly more painful because it rekindles a similar experience as a child when one felt even more helpless and more dependent on the environment.

It is common in social experience to use the expression, "You give me a pain." We have all seen patients who have headache or neck pain and back pain in response to difficult living situations.

The obvious relationship between pain and anxiety is in the patient who has muscle tension in whom one can, indeed, palpate contracted and obviously painful muscles.

Pain, as a symptom, is often seen in individuals who have what psychiatrists call an hysterical character. It must be strongly emphasized that the pain in these patients serves a function. All psychiatric symptoms can be considered as inefficient attempts to deal with conflict. As such, the abrupt removal of the symptomatology must, by necessity, force the patient to look for another avenue

*Delivered at Thayer Hospital, Waterville, Maine, October 26, 1972.

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for the expression of, or hopefully, the solution of the conflict. It is, therefore, important to avoid telling a patient that he has no organic basis for pain without first trying to understand why the patient is experiencing this symptom. A somewhat more psychoanalytic approach to gastrointestinal distress uses such concepts as abdominal pain with organic disease representing unfulfilled early emotional needs. As foreign as that may sound to some physicians, it is my clinical experience that patients in psychiatric treatment often are able to localize this kind of a somatic sensation. One could conceive of resulting hyperacidity and, even more serious, organic pathological disease resulting. Indeed, many kinds of pain do have symbolic meanings and it may be therapeutic for the physician to elicit these.

Another issue in the consideration of pain as another kind of symptom would be in the addict. There are many patients, as we know, in whom there is demonstrable organic disease who have, also, become addicted and present to an emergency room wanting some Demerol®. One should be alerted to this kind of consideration.

A good example of another meaning of pain would be in women with chronic pelvic pain. Writing in 1970, Castelnuovo-Tedesco describes three groups of women of different socioeconomic extraction, some with and some without chronic pelvic pain. These were studied gynecologically and psychiatrically. Regardless of the presence or absence of organic pelvic pathology, pelvic pain patients showed considerable psychopathology clinically and by psychological testing. Many of these patients were eager to undergo a hysterectomy and those who received a hysterectomy generally became pain free. However, they then often substituted for it other symptoms of a psychological nature. The central conclusion was that chronic pelvic pain appears more closely related to the presence of psychiatric disturbance, which is a constant finding, than to the presence of organic pelvic pathology, which is an inconsistent finding. Again, from the psychoanalytic point of view, the wish for the hysterectomy may be an inefficient attempt to deal with some conflict about sexuality or about being a woman which the patient unconsciously feels the removal of the hysterectomy might solve. Obviously, the surgical procedure does not deal directly with the psychological difficulty.

Depression

Depression is a common underlying psychiatric syndrome which often masquerades as physical illness. Some of the "target symptoms" of depression include: weight loss, anorexia, pain, diminished libido, change in sleeping patterns, gastrointestinal disturbance, and many of the psychophysiologic syndromes.

In a study of 35 cases of localized pain without demonstrable organic cause and with depression, Bradley in the *British Journal of Psychiatry,* distinguished two groups: (1) those with chronic pain followed by depression, the depression alone responding to treatment, but with increased tolerance of pain; (2) patients with pain and depression beginning simultaneously both being relieved by treatment of the depression. The author goes on to note that some psychoanalytically oriented writers suggest that pain has a symbolic function and satisfy the depressed patient's need to suffer and as such may protect him from suicidal impulses. This is, I think, an example of the importance of understanding a patient's emotional difficulty before taking steps to relieve overt symptoms when there can be no organic diagnosis made.

Considerations in the Approach to Such Patients

The medical model, which includes the elicitation of the chief complaint, history of the present illness, the past history, the systemic review, and the physical examination and indicated laboratory tests need not be stressed here. In the presence of organic symptoms, however, when no organic cause can be found, the physician is obligated to pursue other avenues. Thomas Szasz, in discussing the problem of pain, talks about a psychiatrist having the task of viewing pain as one would a foreign language and translating it into some kind of meaning for the doctor and for the patient. I would go a step further and suggest that this approach should not be limited to psychiatrically trained physicians but can be of use to all physicians in all specialties. Szasz goes on to suggest that patients feel more comfortable about complaining of dizziness, weakness, palpitation, etc. than about personal unhappiness. The job of the psychiatrist (I would add the physician) is to translate these symptoms for the patient.

Redlich and Freedman call attention to three issues in understanding the patient-doctor relationship: (1) the concept of the sick role and the physician's role; (2) the concept of the participant observer; and (3) the concept of transference and countertransference. In terms of conceptualization of the sick role and the physician's role, it must be remembered that our culture dictates certain kinds of behavior that patients must observe in a medical situation. For many years, there had been an artificial separation of the psyche from the soma and it is the responsibility of the physician to bridge this gap when the patient is not able to. In the work-up of the patient who presents with somatic symptoms in whom there is a chance of an emotional etiology, the physician is aided by acting as a participant observer. We have all experienced emotional reactions to patients such as anger or even the subtle awareness of being relieved when a patient leaves the office or maybe even changes physicians. The perceptive doctor is able to use his own emotional reactions to understand that other people might have similar unpleasant reactions to the patient. Without necessarily informing the patient of his reactions, he may then be able to get some ideas of questions to ask which would relate to an emotional upset. The concept of transference describes reactions that patients may have to a physician which are based historically on other relationships in their lives – such as to parents or siblings. Countertransference refers to a similar kind of situation which

the physician may have in responding to a patient. For example, the physician must be aware that his specific emotional reaction to a patient may relate to his feelings about a loved one and this way may help avoid having his clinical judgement be clouded. This is especially so in the treatment of a patient with an emotional illness.

In the approach to these kinds of patients, it is absolutely indicated to ask a patient directly about what is going on in his life. It may be desirable to try to correlate the timing between the onset of symptoms and what was happening in the patient's life at that time. It is often fruitful to inquire directly about job, marriage, and family history of psychiatric illness. There is no contraindication to inquire about suicidal ideation or intent. This question can be handled very matter of factly and there is no evidence that the physician, by asking about suicide, plants the idea in the patient's mind.

Some group practices and clinics use psychological screening tests; such as, the Minnesota Multiphasic Personality Inventory, as an important adjunct to doing routine physical examinations.

Management

The first decision the physician must make is whether or not to tell the patient that he is, indeed, suffering from a functional illness rather than an organic one. In general, it is desirable to be candid with the patient but to couch this information in terms of being able to offer the patient some help. The way that this is done depends on the patient, his level of sophistication, and even more importantly, on his psychological "need" to preserve the symptom.

The technique of psychotherapy is not limited to psychiatrists. Psychotherapy involves a therapeutic and trusting relationship between the patient and the doctor in which the patient can explore his innermost feelings in confidence and without fear of humiliation. A physician need not delve into unconscious factors and should be very careful about making "wild interpretations" about the determinants of a patient's behavior.

Certainly a physician can be of some aid in directly helping a patient change factors in his environment, such as, his job or his relationship with important people in the community.

There are now a full range of psychiatric medications, which are of significant help in certain syndromes. For example, the group of phenothiazine drugs, particularly Thorazine®, is rather specific for the treatment of schizophrenia. Lithium carbonate has been found useful in the management of manic depressive psychoses when it is begun in the manic phase of the illness. Often these medications are tricky to handle and are not without serious side effects. The physician is advised to study the literature thoroughly before using them and it may even be desirable to consult with a psychiatric physician in terms of dose and indications.

The use of the psychiatric consultation is often a very difficult decision for a physician to make. His own attitudes about psychiatric illness may make it very difficult for him to suggest this. In our clinical experience, however, patients who are suffering from psychiatric problems welcome a psychiatric consultation. The success of the psychiatric consultation is very often dependent on the manner in which it is approached by the patient's personal physician.

Conclusion

Understandably, the major emphasis in the training of most physicians has been one of orientation to somatic symptomatology and search for organic disease. Most physicians do feel much more comfortable once they have been able to make a concrete organic diagnosis. Furthermore, in the absence of being able to make such a diagnosis, many physicians find themselves frustrated and perhaps even feeling helpless.

An attempt has been made here to demonstrate that some of the most common somatic complaints, such as those involving pain, often relate to emotional factors in the absence of any organic syndrome. Of greater importance, is the approach and management of these patients and the desirability of the physician appreciating and being able to communicate to his patient that there is hope and help.

Acknowledgment

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References

Szasz, Thomas S.: "Language and Pain," in Arieti, Silvano (Ed.), American Handbook of Psychiatry, (New York: Basic Books, Inc. 1959).

Rangell, L.: "Psychiatric Aspects of Pain," Psychosom. Med., 15 (1953), pp. 22-37.

Castelnuovo-Tedesco, et al: "Psychosomatic Aspects of Chronic Pelvic Pain," Psychiatry Med., 1 (April, 1970) pp. 109-126.

Bradley, J. J.: "Severe Localized Pain Associated with the Depressive Syndrome," Brit. J. Psychiat., 109 (1963) pp. 741-745.

Redlich, Frederick C.; and Freedman, Daniel X.: The Theory and Practice of Psychiatry, (New York: Basic Books, Inc., 1966).

Singh, G. et al: "Drug Treatment of Chronic Intractable Pain in Patients Referred to a Psychiatric Clinic," J. Indian Med. Assoc., 56 (June 1971) pp. 341-345.

Rose, A. R., M.D.: "Anxiety and Pain," Med. Trial Tech. Q., 17 (1970) pp. 147-155.

Engel, G. L., M.D.: "Psychogenic" Pain and the Pain-Prone Patient. Amer. J. Med., 26, (1959) pp. 899-918.

Huger, Raymond A., M.D.: "Fear, Guilt, Anxiety and Depression in the Painful Man," Arizona Med., 26 (1969) pp. 1030-1031.

Kahn, J. P., M.D.: "How a Psychiatrist Looks at Pain," Med. Times, 98 (Dec. 1970) pp. 127-133.

Bowers, K. S.: "Pain, Anxiety, and Perceived Control," J. Consult. Clin. Psychol., 32, (Oct. 1968) pp. 596-602.

Informed Consent – A Doctor's Dilemma

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Doctors, lawyers, and others informed in the area of health care in the United States have become increasingly aware of a sharp rise in the incidence of medical malpractice cases in this country. Much has been written about the psychological and sociological reasons for the awakening litigiousness of recipients of health care. Courts and lawyers have made many attempts to analyze the various legal facets of this trend in the law of malpractice litigation.

In recent decades, the legal doctrine of negligence has generally been the theory of law utilized as a foundation in malpractice cases. This doctrine imposes a duty upon all persons to conform to certain objective standards in order to prevent the creation of unreasonable risks of harm to others. Thus, a failure on the part of any person to conform to such objective standards, normally referred to as the "reasonable man" standard, means negligence on the part of the actor and an obligation to respond in civil damages to anyone injured thereby.[1]

In those malpractice cases alleging negligence because of a bad result from a medical or surgical procedure, the almost classical defense urged by doctors has been a response that the complication which occurred was one which took place with sufficient frequency so that its mere happening did not spell negligence. For example, a thyroidectomy was performed on a 43-year-old woman. The surgeon failed to inform the patient that an ultimate effect of the operation was the possibility of damage to the recurrent laryngeal nerves resulting in loss of voice. As a result of the operation which was "consented" to by the patient, she was unable to speak above a whisper. She brought suit alleging that the surgeon was negligent in his selection of the surgical procedure. Testimony established that, regardless of which technique was employed, statistics showed a 2 percent incidence of permanent damage to the laryngeal nerves, even in the absence of negligence.[2] The argument flowing from this that even a reasonably well-trained doctor exercising the usual degree of skill in his area of practice could not necessarily have prevented the unfortunate result from occurring.

For many years, a response of this nature ruled the day and got the doctor out of the court room and back to the operating room. Within the past ten years, however, there has developed a counter-response to such an argument via the utilization and modification of the basic legal doctrine of trespass to the person or, as it is commonly known, "battery." Plaintiffs, on many occasions, responded that if the untoward result was a complication which could arise from a procedure, even in the absence of negligence by the physician, the patient should have been informed of the possibility of such a risk, which must have been known to the doctor, so that he could give his consent based upon an understanding that the result could happen despite the exercise of proper medical care on the part of the physician.

In *Natanson v. Kline,*[3] for example, the non-negligent administration of cobalt radiation treatment caused serious injuries. This was the type of injury which sometimes happened with such an inherently dangerous treatment. The physician had failed to warn of the risk and the jury was permitted to infer from the evidence that if the patient had been warned, she would have declined the treatment.

It is this change in emphasis in the area of consent which has created for the surgeon the dilemma of a two-edged scalpel. The edges of the scalpel are: (1) When such a complication occurs, it is so rare in incidence that it immediately indicates negligence on the part of the surgeon, or (2) it occurs with sufficient frequency so that its mere happening cannot permit an inference of negligence. If the surgeon opts to operate with the second edge of the scalpel, he must be prepared to prove that the patient was apprised of the possibility of such a complication before consenting to the operation.

An understanding of the basic theories of "negligence" and "battery" is necessary to fathom this dilemma since the two theories become more and more enmeshed in the evolving jurisprudence of the malpractice field. Traditionally, the cause of action known as negligence is made up of the following components:

1. A duty, or obligation, recognized by the law, requiring the actor to conform to a certain standard of conduct, for the protection of others against unreasonable risks.

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2. A failure on his part to conform to the standard required.
3. A reasonable close causal connection between the conduct and the resulting injury. This is what is commonly known as "legal cause," or "proximate cause."
4. Actual loss or damage resulting to the interests of another.[4]

Negligence is characterized as an unintentional tort whereby the actor does not, in fact, intend to commit the act which causes the injury. Thus, if a doctor intends to perform a certain procedure and performs the procedure without the consent of the patient, a strict interpretation of the doctrine would indicate that he is not negligent so long as he performed the procedure properly. He has, however, committed a battery. If, however, the doctor has not secured the consent of the patient and performs the procedure in a manner below the standard of care required, he has committed both the intentional tort of battery and the unintentional tort of negligence.

Battery is generally defined as an intentional unpermitted contact with another person. It makes no difference whether or not the intent to touch is motivated by hostility, since the key factor is the absence of consent to the contact by the plaintiff.[5] In the medical field, the application of this theory of law is succinctly stated as follows:

> [E]very individual has a right to the inviolability of his person which forbids a surgeon or physician to invade the bodily integrity of his person. Whenever a surgeon or physician, without the patient's permission, performs an operation or renders medical treatment, he prima facie commits a battery.[6]

This concept of consent has always been the keystone in the definition of the legal term "battery." However, in recent times the courts have modified the traditional concept of consent, as far as the field of medicine is concerned, by attaching a modifier to the word "consent" and have created the doctrine of "informed consent." This doctrine is one whereby a consent effective to negate an act of battery can be secured only when the patient has an *understanding* of the procedure, alternatives available, and risks involved.[7] This same name is also applied to another doctrine utilized by courts in imposing a duty upon doctors to communicate information to patients relating to the inherent and potential hazards of the procedure, alternatives and results likely if the patient remains untreated.[8]

Regrettably, this doctrine of "informed consent" has become somewhat confused. This confusion results, to some extent, from an attempt by courts to circumvent a short statute of limitations for battery by finding an additional duty sounding in negligence to reach a longer statute of limitations and thus give the plaintiff his day in court. One can readily see that a doctor should secure the "informed consent" of the patient in order to have the requisite permission necessary to perform the procedure. Consent negates a trespass or battery. Some courts have said a doctor must supply such information in order to fulfill another separate duty to the patient.

> True consent to what happens to one's self is the informed exercise of a choice, and that entails an opportunity to evaluate knowledgeably the options available and the risks attendant upon each. The average patient has little or no understanding of the medical arts, and ordinarily has only his physician to whom he can look for enlightenment with which to reach an intelligent decision. From these almost axiomatic considerations, springs the need, and in turn, the requirement, of a reasonable divulgence by physician to patient to make such a decision possible.[9]

The creation of this second duty for procedural reasons is really a restatement of the basic duty of securing "informed consent" from a different perspective.

Perhaps the best explanation that can be made of the distinction between the two doctrines of "informed consent" was made in 1972 in the case of *Canterbury v. Spence*.[10] The court indicated that consent to a touching is predicated upon the patient's understanding of the procedure, etc., while the doctor's duty to disclose focuses "upon the nature and content of the physician's divulgence rather than the patient's understanding or consent."[11] The court went on to say that

> Adequate disclosure and informed consent are, of course, two sides of the same coin – the former a *sine qua non* of the latter. But the vital inquiry or duty to disclose relates to the physician's performance of an obligation, while one of the difficulties with analysis in terms of "informed consent" is its tendency to imply that what is decisive is the degree of the patient's comprehension.[12]

Regardless of whether "informed consent" is viewed from the perspective of the patient's understanding or from the perspective of the physician's efforts to convey enough information upon which to make an intelligent decision, the most troublesome area for doctors and lawyers alike is the determination of the level or degree of information which must be given to a patient in order that the consent be truly "informed." Many courts have ruled that the medical standards of the community relating to disclosure of particular facts govern what information must be given to a patient.[13] Other courts have said that the duty results from a "fiduciary relationship" requiring the utmost of good faith in informing the patient.[14]

His duty of disclosure extends beyond the realm of *risks*. He must disclose to his patient all material *facts* which reasonably should be known if his patient is to make an informed and intelligent decision.[15]

The *Hunter* Court, for example, said that it was irrelevant whether the failure to disclose a risk of potential complication was willful or merely negligent since the

true wrong was a breach of another duty, the fiduciary duty to disclose.

These seemingly irreconcilable duties begin to form a more logical pattern when one analyzes the various types of facts required to be disclosed by the physician. For purposes of analysis, factual disclosure can be placed in categories of "known consequences," "probable consequences," and "complications." Except in circumstances constituting an emergency,[16] or when disclosure would cause substantial detriment to the patient,[17] disclosure of "known consequences" is required in all cases. Examples of known consequences include scarring resulting from the stripping of varicose veins;[18] sterilization as the result of a transurethral prostatic resection;[19] shortening of a leg as the result of the use of a metal prosthesis to treat a fractured femur;[20] and reduction in breast size as the result of mammoplasty.[21]

"Possible consequences" includes that group of occurrences which are not certain to happen as the result of a procedure but have such an incidence level of occurrence that the physician can reasonably expect that such might occur. Into this area falls cases such as the possibility of incontinence and impotence following G. U. surgery,[22] or the possibility that an incision will fail to heal properly due to the previous existence of Hodgkins disease.[23]

Courts, in determining a physician's liability in the foregoing situations, generally allow the jury to decide the issue on the basis of whether the patient would have consented to the operation had he or she been advised of the consequences.[24]

This subjective method of determining causality recently came under attack in *Canterbury v. Spence,*[25] in which the court opted for an "objective" test. The court felt that reaching a decision on the basis of what the specific patient would have done had he been fully informed would be based on an "assessment of the patient's credibility" in answering a question which "hardly represents more than a guess, perhaps tinged by the circumstance that the uncommunicated hazard has in fact materialized."[26]

The *Canterbury* Court said that such a subjective test placed the physician "in jeopardy of the patient's hindsight and bitterness."[27] The court went on to declare that the causality issue should be resolved

> . . . on an objective basis: in terms of what a prudent person in the patient's position would have decided if suitably informed of all perils bearing significance. If adequate disclosure could reasonably be expected to have caused that person to decline the treatment because of the revelation of the kind of risk or danger that resulted in harm, causation is shown, but otherwise not.[28]

Regardless of whether the subjective or objective standard of determining causality is utilized, courts will probably not require the testimony of expert medical witnesses unless some question arises about the medical impact of disclosure upon the health of the patient, etc. "Experts are unnecessary to a showing of the materiality of a risk to a patient's decision on treatment, or to the reasonably, expectable effect of risk disclosure on the decision."[29]

"Complications" is the most nebulous category in the area of "informed consent." Complications are those events which result from a medical procedure which are neither intentionally caused, nor desirable, nor anticipated. They are the untoward, unexpected results of medical procedures. The question still remains, however, whether such an event was a recognized risk at the time the operation was performed. Some courts have said that the practice of the local medical profession will determine whether the disclosure is required and that the custom of the medical profession to warn must be established by expert medical testimony.[30]

> This approach clouds the issue. Even if the local profession does not inform of certain risks, this has only a derivative relevance on the question of whether the risk is so likely to occur that a patient should be informed of it. . . . [I]t is quite clear that an industry cannot by concurring in substandard practices set their own standard of care.[31]

Recently, however, courts have adopted the position of the treatise writers in holding that doctors may not, among themselves, determine what facts must be disclosed. In *Cooper v. Roberts,*[32] for example, the plaintiff underwent a gastroscopic examination after signing a blanket consent form. As a result of the examination, her stomach was punctured by the fiberscope used in the examination. "The fiberscope was considered an improvement over its forerunner, the semi-rigid scope, in terms of comfort for the patient, visibility of the area under examination, and safety. The incidence of perforation with the semi-rigid scope was approximately 1 in 2500, or .0004 percent."[33] The patient had not been informed of any collateral risks of perforation or otherwise. The appeals court, in overruling the trial judge's charge to the jury that the duty to disclose must be measured in light of the amount of disclosure which would be made by a reasonable practitioner in the community, stated that:

> As the patient must bear the expense, pain and suffering of any injury from medical treatment, his right to know all material facts pertaining to the proposed treatment cannot be dependent upon the self-imposed standards of the medical profession.[34]

The question of when a risk is substantial enough to require disclosure to the patient is one that cannot be easily answered. Disclosure has been required where there was a 3 percent chance of death, paralysis, or other injury,[35] and where there was a 1 percent chance of hearing loss.[36] Disclosure has not been required where there was a 1/800,000 chance of aplastic anemia,[37] or a 1/250 to 1/500 chance of perforation of the esophagus.[38]

> There is no bright line separating the significant from the insignificant; the answer in any

case must abide a rule of reason. Some dangers – infection, for example – are inherent in any operation; there is no obligation to communicate those of which persons of average sophistication are aware. Even more clearly, the physician bears no responsibility for discussion of hazards the patient has already discovered, or those having no apparent materiality to patient's decision on therapy. The disclosure doctrine, like others marking lines between permissible and impermissible behavior in medical practice, is in essence a requirement of conduct prudent under the circumstances.[39]

This "prudent man" requirement of disclosure causes a dilemma to the physician when a complication occurs. On the one hand he argues that he acquired informed consent without telling the patient of the probable complication while, at the same time, defending the negligence action by saying that such a complication often occurs without negligence. If the physician can argue that the complication has been known to occur without negligence, he should be aware of the probability of such complication to the extent that he should have informed the patient, prior to the procedure, of the likelihood of such a complication. Thus, if attorneys bringing malpractice actions were to combine a count for battery with a count for negligence, it would seem that they would have the doctor riding a black horse in one direction at the same speed that he is attempting to ride a white horse in the other direction.

How then do doctors protect themselves from the horns of this dilemma? It appears to be clearly established that the doctors must live up to an acceptable standard of disclosure of potential risks of complications and should secure evidence of such disclosure through appropriate "informed consent" forms. As is the case in most, if not all, negligence actions, the reasonable man standard against which the actor's conduct will be measured is frequently uncertain until the jury, acting in its wisdom, determines what that standard should have been in the particular situation.

In the situation of informed consent involving complications, the jury must make this determination on the basis of expert medical testimony or, depending upon the jurisdiction, upon the basis of whether it was reasonable to expect that the patient would attach significance to the undisclosed fact. Regardless of which approach the jury takes, expert medical testimony concerning the general standards of disclosure with respect to medical complications can be helpful, if not crucial. While a court may not accept a defensive set of standards, reasonableness dictates acceptance of standards developed in good faith and adhered to by the medical community.

The answer for the practitioner then becomes more definable. Doctors and surgeons should organize themselves into appropriate peer groups to determine in advance what the standards of disclosure should be with respect to various medical and surgical procedures. A checklist created by the peer group should be utilized by all physicians practicing within the area to establish that they have, in fact, lived up to the level of disclosure set by the appropriate peer group. This checklist should be constantly reviewed to insure that it accurately reflects the latest medical knowledge concerning the procedure. In fact, such a checklist could easily be incorporated into the appropriate consent form so as to provide evidence of the fact that appropriate disclosure was in fact made. This procedure in some jurisdictions will not, however, totally dispel any possibility of legal liability. It does, however, go far to show an attempt by the practitioner to conduct himself in a manner which manifests action which is reasonable under the circumstances. The solution appears obvious but also appears to have been rarely accomplished. The day of the "doctor knows best" feeling of patients is waning as the breakdown of the inter-personal relationship between the doctor and his patient increases. Doctors must, for their own protection, come to realize that "it is the prerogative of the patient, not the physician, to determine for himself the direction in which his interests seem to lie."[40]

References

1. *Wm. Prosser, Law of Torts* § 30 at 143 (4th ed. 1971).
2. DeFillipo v. Preston, 53 Del. 539, 173 A.2d 333 (1961).
3. 187 Kan. 186, 354 P.2d 670 (1960).
4. *Wm. Prosser, Law of Torts,* § 30 at 143 (4th ed. 1971).
5. *Id. at* § 9.
6. McCoid, *A Reappraisal of Liability for Unauthorized Medical Treatment,* 41 Minn. L. Rev. 381, 392 (1957).
7. *See, e.g.,* Waltz & Scheuneman, *Informed Consent to Therapy,* 64 Nw. U. L. Rev. 628, 629 (1970).
8. *Id.* at 644.
9. Canterbury v. Spence, 464 F.2d 772, 780 (D.C. Cir. 1972).
10. *Id.*
11. *Id.* at 780, n.15.
12. *Id.*
13. *See e.g.,* DeFillipo v. Preston, 53 Del. 539, 550, 173 A.2d 333, 339 (1961); Haggerty v. McCarthy, 344 Mass. 136, 141, 181 N.E.2d 562, 565, 566 (1962); Roberts v. Young, 369 Mich. 133, 139, 119 N.W.2d 627, 630 (1963); and Aiken v. Clary, 396 S.W.2d 668, 675 (Mo. 1965).
14. Hunter v. Brown, 4 Wash. App. 899, 484 P.2d 1162 (1971).
15. *Id.* at 1166.
16. *E.g.,* Dunham v. Wright, 423 F.2d 940, 941, 942 (3d Cir. 1970); Wells v. McGehee, 39 So.2d 196 (La. App. 1949); Jackovach v. Yocom, 212 Iowa 914, 237 N.W. 444 (1931).
17. Roberts v. Wood, 206 F. Supp. 579, 583 (D. Ala. 1962); Woods v. Brumlop, 71 N.M. 221, 377 P.2d 520, 525 (1962); Waltz & Scheuneman, *Informed Consent to Therapy,* 64 Nw. U. L. Rev. 628, 641-43 (1970).
18. Govin v. Hunter, 374 P.2d 421 (Wy. 1962).
19. Bang v. Charles T. Miller Hospital, 251 Minn. 427, 88 N.W.2d 186 (1958).
20. Russell v. Harwick, 166 So.2d 904 (Fla. 1964).
21. 16 *The Citation* 81 (1967).
22. Fontenelle v. United States, 327 F. Supp. 801 (D. N.Y. 1971).
23. Neeriemer v. Superior Court of Maricopa County, 13 Ariz. App. 460, 477 P.2d 746 (1970).
24. Plante, *An Analysis of "Informed Consent,"* 36 Fordham L. Rev. 639, 666-67 (1968) Waltz & Scheuneman, *Informed Consent to Therapy,* 64 Nw. U.L. Rev. 628, 646-48 (1970); Comment, *Informed Consent in Medical Malpractice,* 55 Calif. L. Rev. 1396, 1411-14 (1967).
25. 464 F.2d 772, 790 (D.C. Cir. 1972).
26. *Id.*
27. 464 F.2d at 791.

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The Physician Assistant in Maine: Two Pending Proposals

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Addendum

Since submission of this manuscript, LD 1088 and 1360 have been significantly altered by amendments. As before, LD 1088 requires that PA's working with MD's must be graduates of programs approved by the Board of Registration of Medicine. But, LD 1088 now permits physician assistants to work with osteopaths if the PA's training programs are approved by the Board of Osteopathic Examination and Registration. The bill now exempts nurse practitioners from its coverage if their training programs are approved under LD 1360's revised provisions which now stipulate joint approval of nurse practitioners by both the boards of nursing and medicine. LD 1088 has excluded optometric procedures from the purview of physician assistants. The Maine Medical Association is backing both 1088 and 1360 as amended. These changes greatly improve both 1088 and 1360 so that they are now politically feasible bills. LD 498 has not been further revised and hence still contains the several ambiguities and problems mentioned in our paper.

Both LD 1088 and 1360 as amended are predominantly delegatory bills but have some regulatory authority elements which can protect the public interest. Because we feel that the passage of some sort of legislation is urgent, we feel that they can and should be passed.

The phenomenon of the physician assistant is a recent one not only in Maine but in the rest of the nation as well. The formal training and employment of such persons was an outgrowth of the public clamor for more and better health services in the 1960s. Many groups, including the Carnegie Commission,[1] proclaimed that part of the country's problems in health care were due to inadequate manpower. The Federal Government adopted this view and provided extensive federal funding for training physician assistants in the late 1960s.

Over half of the United States have passed some sort of regulatory legislation to deal with physician assistants. Here in Maine our Legislature is currently considering two proposals regarding them. While both regulate and permit the use of physician assistants and must be considered an improvement over the present uncertainty as to their legal status, the two bills vary considerably in their treatment of the regulatory problem. LD 1088, titled "An Act Relating To Physician Assistants," is primarily a so-called "delegatory" statute relying on the deterrent effect of the rules of liability which it creates and on training program approval to guide the use of physician assistants into proper channels. The other Bill LD 498, titled "An Act Relating To The Sanction And Conduct of Assistants To Physicians," is classified as a "regulatory authority" act which provides an ongoing supervisory authority to monitor and guide the use of physician assistants in Maine. It is the authors' opinion that the latter type of statute is preferable but that LD 498 as proposed would benefit from modifying amendments.

Nursing groups have submitted a 3d statute to the current legislature, LD 1360. However, this statute simply says that RN's may be serving as physician assistants. It does not define or regulate their role in anyway and essentially relies on custom and usage as we currently do in Maine. It is compatible with but does not substitute for either LD 498 or LD 1088.

We must distinguish the physician assistant, and we include nurse practitioners, from the so-called medical assistant. A physician assistant does tasks which are usually performed by a physician, as opposed to secretarial or nursing type tasks. Thus, a physician assistant is different from an office nurse, a secretary, laboratory technician or any combination of such personnel.

Even more crucial is the distinction between a physician assistant and an independent medical practitioner. As the term implies, a physician assistant works under and is responsible to an employing physician. That physician assumes liability for all acts which the physician assistant may perform for him. If legislative control of the physician assistant is inadequate, then there is a real danger that the physician assistants may become independent practitioners.

Seven years ago Duke University started a two-year training program.[2] The training, predominantly within the University, was about half didactic and half clinical and has been criticized as a "mini-medical school" for teaching irrelevant didactic material. Duke initially drew on the large supply of military corpsmen but has more recently expanded to include students with many different types of medical backgrounds.

The second major training model are the MEDEX Programs.[3] Former military corpsmen and other persons with extensive previous medical training receive a brief (two to four months) didactic training course and then are sent out for eleven to fourteen months on-the-job training in a preceptorship. Through these preceptorships with potential subsequent employers, the MEDEX Pro-

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grams, unlike the Duke program, can influence deployment to areas of need. In this way the MEDEX programs have directed their graduates predominantly toward rural general practitioners, discouraging the association of physician assistants with secondary care specialist and encouraging them to emigrate from medical centers.

The nurse practitioner programs are still another basic type of physician assistant training program in this country. The program started in Colorado is the prototype.[4] The mixture of on-the-job training versus didactic emphasis varies from program to program but generally the former is predominant. Control over the deployment of such trained personnel as in MEDEX type programs is possible.

Here in Maine we have three programs that are formally training physicians' assistants. All three use nurses. They are the Pediatric Nurse Associate Training Program, The Family Nurse Associate Training Program and a new mid-wives training program all based in Portland. In addition, the Dartmouth MEDEX Program sends many students to Maine for preceptorships and subsequent employment.

Currently there are over fifty physician assistant training programs which have already graduated students in the United States. There are more than an equal number of training programs in the early stages of development. The total number of physician assistant graduates in the country is probably over 400; there are about 40 graduates or students in training in the State of Maine now.

The American Medical Association and the Joint Commission for Hospital Accreditation have both strongly endorsed the physician assistant concept. Nationally two groups are attempting to assure adequate training for physician assistants. The AMA has developed an accreditation system for training programs and the National Board of Medical Examiners is developing a board type examination to be administered to graduates of physician assistant programs.[5] Both groups are concerned with the assistant to the primary care physician. Money for training programs has come from several federal sources in the past; but currently it is consolidated in the Bureau of Health Manpower of the National Institutes of Health. Most physician assistant training programs get some of their funds from this office.

After training, physician assistants are assigned to such tasks as general or screening histories and physicals, frequent manual tasks like laceration suturing and caring for common problems which can be formally or informally summarized in a protocol. In order to improve the number and quality of services which a practice unit provides, a physician assistant must perform a significant number of complete service skills so that the supervising physician does not have to see every single patient. The corollary of this is that the physician assistant must be capable of providing medical care safely and competently without the direct or immediate supervision of the physician.

There is no one way in which the physicians supervise physician assistants. The basic premise is that the physician supervises as much as necessary to provide equivalent quality service because he is legally liable for that service. Physicians usually review charts and rely on the physician assistant to request more immediate help when he discovers he is beyond his capabilities. The physician assistant is usually expected to follow a written or, more commonly, an unwritten protocol which he has developed with his supervising physicians.

Physician assistants are generally paid by the hiring institution or practitioner, usually on a salary basis. Their services are generally charged to the patients by their employer at the usual and customary charge for that employer. The presumption is that the supervising physician assures a quality of service equal to that which he himself would provide. He is also liable. Therefore, the charge is the same.

One practical advantage of permitting and regulating the use of physician assistant manpower is that they are already on the medical care scene and providing health services. Physician assistants do increase the number of health services in medical practices.[6] They can increase the quality of care given if the physicians delegate common problems and free themselves to concentrate on the more difficult cases to which the bulk of their physician training has been directed. Physician assistants can generally make medical care more available and accessible to patients; they can help the physician to expand office hours, to provide extra-office services and home visits. To the extent the physician assistants are drawn from previously wasted manpower sources, such as ex-military corpsmen and retired nurses, then physician assistant programs salvage otherwise wasted manpower and training.

Another political reality here in Maine is organized nursing's apparent concern about the Board of Registration in Medicine approving nurse practitioners without major input from the Nursing Board.[7]

The task of the proposed legislation is to permit the growth of the physician assistant movement to the extent it has merit and fills the need for medical care delivery, while at the same time protecting the public interest by assuring the quality of care. The two bills pending before the Maine Legislature begin their approach to this formidable undertaking with several similarities. Both recognize and sanction the physician assistant. The physician assistant, properly employed, is thereby protected from the charge of illegal practice of medicine. It has occasionally been contended that the physician assistant is already so protected by the shield of prior custom and usage. Limited delegation of duties to unlicensed helpers is not new. Many assume that personal over-the-shoulder supervision of at least those tasks that did not present unusual difficulty is sanctioned by custom within the medical community, even if those tasks do constitute "treatment" within the meaning of the Medical Practice Act. Unfortunately, the courts of many states have never had to pass on that point; and therefore in those states it

is not definitely settled. Even more uncertainty exists in more attenuated cases where the physician is not present and actively supervising. Further, there are occasional court decisions which cast doubt on the wisdom and safety of reliance on custom. The State of California, prior to enactment of its physician assistant act, successfully prosecuted a surgical technician for the illegal practice of medicine where at the direction and in the presence of the surgeon he operated a cranial saw.[8] The physician was also convicted as an aider and abettor. Another California physician was held liable for employing an unlicensed person (a foreign medical graduate) to administer an anesthetic despite the physician's personal supervision and despite the fact that the unlicensed person was highly skilled and experienced.[9] And an occasional court has permitted a defendant's lack of licensure or other formal authority to stand as presumptive evidence of failure to possess the requisite skill and competence.[10] The desirability of clarification of this point by statute is obvious.

Another similarity between the two bills is their treatment of the requirement of the physician's personal presence. Neither bill requires presence. This provision is especially important in a thinly populated, medically underserved area like Maine. This provision is of special interest when read with the stipulation also common to both bills, that physician presence is required where necessary to maintain the level of patient care as high as that provided by the physician. In addition, both bills expressly declare that the physician shall be legally liable for the physician assistant's activities and that the relationship between them is that of principal and agent. That declaration adds nothing to prior law but does serve to make clear that it is the legislature's intent, in creating this new health professional free from the physician's presence, to make him a dependent provider of health care not an independent one. This dependent role for physician assistants results in the legally rather unusual requirement that the assistant be held to the same standard of performance as a physician. Were this not done however, there would be a falling off of the quality standard. Both bills seek to avoid that happening by reviving the presence requirement in any case where the absence of the physician results in a lessening of the quality of care.

That provision should tend to promote rigorous training and careful delegation and supervision of duties. One less obvious consequence, however, is the imposition of something like an insurance element on top of the duty to exercise care. For example, a physician assistant in the course of performing minor treatment which he is qualified to do and which he does competently, sees but does not appreciate the significance of another more serious condition which is entirely beyond his training but within the training and experience of a physician. Both bills would hold the physician liable. The same result would follow where the physician assistant in the course of competently treating a minor condition is confronted by a sudden emergency to which he responds to his best skill and judgment which is, nevertheless, inferior to the skill and judgment which a physician would bring to the same crisis.

These two safeguards, liability plus the limited presence requirement, should promote proper effective use of the physician assistant. This approach is preferable to an undeviating insistence on the presence requirement which would unduly hamper maximal use of physician assistants.

A further point of similarity of the two bills is their requirement that PAs be graduates of an approved training program.

The major difference between the two bills is that LD 498 undertakes to create an ongoing regulatory scheme. The other bill proposes nothing on this topic but relies on training program approval and on the self-policing to be expected from the rules of liability which it enacts. Unfortunately, there is, as yet, no data on the effectiveness of such controls. Statistically, two-thirds of the states with physician assistants legislation have chosen the "regulatory authority" statute than have relied on delegatory laws.

LD 498 proposes a four-point control system to be vested in the Department of Health and Welfare: (1) power to specify which physicians will be permitted to employ physician assistants; (2) approval of training programs; (3) a registry system for approved physician assistants; and (4) power to classify activities as non-delegable. Perhaps the Board of Registration in Medicine rather than the Department should be made the supervisory authority. There are two reasons for this. First, a great amount of definitional work must be done in spelling out what training programs are to be approved and what tasks are to be considered non-delegable, and here the expertise of a professional board seems desirable. Second, the bill recognizes that control of employer physicians is properly a function of the professional board. The result is a dual agency approach, giving part of the supervisory authority to the Department of Health & Welfare and part to the Board of Registration in Medicine. A single supervisory authority would be infinitely simpler.

The part of the statute which calls for a registry system contains at least three present problems. First, it lacks an express statement of grounds for revocation of authority to practice as a physician assistant. This feature is found in the statutes of several other states having physician assistant legislation. LD 498 calls for annual reregistration but sets no standards either for initial registration or reregistration except that the candidate be a graduate of an approved training program and be "otherwise approved by the Department." That last phrase invites constitutional attack for vagueness. The second problem comes from the wording of the clause entitling presently licensed health professionals such as nurses, physical therapists and others to register as physician assistants without payment of the fee required of other applicants. It is possible to read that sentence as also excusing those licensed individuals from the training requirement. The statement of fact appended to the LD reinforces this im-

pression by stating "other health care professionals . . . would automatically be determined as appropriate persons to whom physicians may delegate certain tasks under supervision." Probably exception from physician assistant training programs is not intended and one hopes that this will be clarified. The third and most serious drawback is the extremely broad interpretation of "physician assistant" implied in the statement of fact. This implication is that the bill would include other health personnel such as "medical technicians, X-ray technologists, surgical technicians, and the like" as physician assistants. The bill itself does not define physician assistants. The automatic approval of such technicians, whose training typically does not prepare them to perform the physician assistant's duties as defined earlier in this paper, will greatly weaken the bill.

The power to declare activities non-delegable is a desirable control and the draftsman wisely allows for exercise of this power on an individual basis. This permits recognition of subspecialties within the ranks of the physician assistant. However, in order to recognize the benefit of this control, the list of non-delegable duties will have to be painstakingly derived and painstakingly applied to individual cases. This is one area in which a professional supervisory board may be able to move with more expertness and economy than a department of the state government.

LD 498 takes realistic account of the prerogatives of other health professionals. It grants osteopaths the same chance to employ assistants as MDs and subjects them to the same restrictions. It prohibits the delegation of activities usually performed by optometrists, dentists, psychiarists and pharmacists. It provides for some input from the nursing profession via its advisory board. This advisory board might be more acceptable and affective if its membership were amended to include representatives from the nurse practitioner training programs in the state, practicing PA's and perhaps others. The failure of LD 1088 to make similar concessions may seriously hinder its chance of passage.

Both LD 498 and LD 1088 rank as important enabling legislation in a field sufficiently complex and obscure to require statutory treatment. Reliance upon custom and usage does not seem justified. Another traditional alternative is that of licensure. A specific designation of training requirements and usually a specific job description, licensure theoretically assures quality. However, experience with licensure in other health fields indicates that little guarantee can be made.[11] Because it is so explicit, licensure stymies experimentation with and utilization of a new manpower source.

Of the two types of statutes represented by the two LDs, the regulatory authority approach of LD 498 gives more assurance of protection of the public interest. Objections can be found to this bill as presently drawn, but these are an argument for amendment and improvement, not an excuse for summarily rejecting it. The medical practice of this State will be well served by thoughtful legislative evaluation and upgrading of this proposal.

References

1. Higher education and the nation's health, a special report and recommendations by the Carnegie Commission on higher education. Ocotber 1970, McGraw Hill.
2. Stead, E. A.: At Duke: a new approach to the doctor shortage. Resident Physician 13: 84-96, 1967.
3. Smith, R. A., Bassett, J. R., Markarian, C. A. et al: A strategy for health manpower-reflections on an experiment called MEDEX. JAMA 217: 1362-1367, 1971.
4. Silver, H. K., Ford, L. C., Day, L. R.: The pediatric nurse practitioner program. JAMA 204: 298-302, 1968.
5. Todd, M. C.: National certification of physicians' assistants by uniform examinations. JAMA 222: 563-566, 1972.
6. Smith, R. A., Anderson, J. R., Okimoto, J. T.: Increasing physician productivity and the hospitalization characteristics of practices using Medex. Northwest Med. 72: 701-706, 1971.
7. The conflict between nurses and physicians is very similar to that which occurred in 1971 in New York. Sadler, A. M., Sadler, B. L., Bliss, A. A.: The Physician's assistant today and tomorrow. New Haven: Yale University Press. 1972, pp. 120-132.
8. Whittaker v. Superior Court of Shasta County, 438 P.2d 358 (Cal. 1968).
9. Magit v. Board of Medical Examiners, 57 Cal. 2d 74, 366 P.2d 816 (1961).
10. Note, Tort Liability & The California Health Care Assistant, 45 So.Cal.L.Rev. 768.
11. Kessel, R.: The AMA and the supply of physicians. Health Care: part I, Law and Contemporary Problems (Duke), Spring, 1970.

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28. *Id.*
29. 464 F.2d at 792. See also Waltz & Scheuneman, *supra,* note 22 at 635. "A risk is thus material when a reasonable person, in what the physician knows or should know to be the patient's position, would be likely to attach significance to the risk or cluster of risks in deciding whether or not to forego the proposed therapy." Waltz & Scheuneman, *supra,* at 640.
30. *See, e.g.,* DeFillipo v. Preston, 53 Del. 539, 550, 173 A.2d 333, 339 (1961); Haggerty v. McCarthy, 344 Mass. 136, 141, 181 N.E.2d 562, 565-66 (1962); Roberts v. Young, 369 Mich. 133, 139, 119 N.W.2d 627, 630 (1963); Aiken v. Clary, 396 S.W.2d 668, 675-76 (Mo. 1965).
31. Smith, *Battery in Medical Torts,* 16 Clev.–Mar. L. Rev. 22, 36 (1967).
32. 220 Pa. Super. 260, 286 A.2d 647 (1971).
33. 286 A.2d at 648.
34. *Id.* at 650.
35. Bowers v. Talmadge, 159 So.2d 888 (Fla. App. 1963).
36. Scott v. Wilson, 396 S.W.2d 532 (Tex. Civ. App. 1965).
37. Stottlemire v. Cawood, 213 F. Supp. 897, 898 (D.C.C. 1963), *new trial denied,* 215 F. Supp. 266 (1963).
38. Starnes v. Taylor, 272 N.C. 386, 393, 158 S.E.2d 339, 344 (1968).
39. Canterbury v. Spence, 464 F. Supp. 772, 788 (1972).
40. *Id.* at 781.

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Suppressed Plasma Renin Activity and Primary Aldosteronism

Paul I. Jagger, M.D.*

The appearance in the 1960's of a relatively simple radioimmunoassay for plasma renin activity (PRA) has resulted in the rapid proliferation of literature relevant to renin changes in hypertension. At the same time, this radioimmunoassay has made the measurement of PRA available for general clinical use and, therefore, the physician who sees patients with hypertension needs to understand the significance of this recent literature in order to know when to utilize this tool. Renin, of course, is the enzyme produced in the cells of the juxtaglomerular apparatus of the kidney that acts on a circulating substrate to initiate the formation of angiotensin II. Angiotensin II is both a potent vasoconstrictor and a stimulus to aldosterone secretion by the adrenal cortex. The observation concerning renin that has received the most attention recently is the somewhat unexpected finding that as many as 25% of patients with hypertension have suppressed peripheral PRA. This means that PRA remains low even after the patient has been subjected to a stimulus that would increase PRA in the normal individual. The standard stimulus usually employed involves placing the patient on a low sodium diet for 3-5 days and then having him ambulate for 2 hours. Suppressed PRA has been emphasized in the past as a critical finding in patients with primary aldosteronism. Though this is true, it now has been shown that the majority of patients with suppressed PRA do not have aldosterone hypersecretion and an alternate explanation for suppression is needed in these cases.

Primary Aldosteronism

To review the situation in primary aldosteronism, autonomous hypersecretion of aldosterone by a lesion in the adrenal cortex, classically, by an adenoma, results in increased sodium reabsorption by the distal renal tubule, leading to extracellular fluid volume expansion and hypertension. This expansion is "sensed" by the juxtaglomerular apparatus cells and their production and secretion of renin are suppressed. The fall in renin and, subsequently, angiotensin II levels, however, does not result in decreased aldosterone secretion as it does in the normal, for, as noted, the aldosterone secretion is autonomous. It is the combination of the suppressed peripheral PRA and the fixed high aldosterone level that confirms the diagnosis of primary aldosteronism. Very rarely, an adrenal tumor can produce the precursor 11-deoxycorticosterone (DOC) instead of aldosterone and in these instances aldosterone levels will be low, but DOC levels will be elevated. Since aldosterone stimulated reabsorption of sodium from the distal tubule takes place mainly in exchange for potassium, hypokalemia becomes a prominent feature in most patients with primary aldosteronism and the measurement of serum potassium provides a simple screening test.

Other Documented Adrenal Cortical Diseases

Peripheral PRA also is suppressed in two other well defined situations where there is documented steroid hormone excess, sodium retention, and hypertension, namely, Cushing's syndrome and certain forms of the adrenogenital syndrome. In Cushing's syndrome, the salt retaining effect of large amounts of glucocorticoids offers an adequate explanation for the suppression of PRA, however, increased production of mineralocorticoids also has been found in some patients. In the C-11 hydroxylase deficiency form of the adrenogenital syndrome, there is increased production of androgens and of DOC and it is the DOC that causes sodium retention and PRA suppression. These patients present as masculinized females or precocious males with hypertension. In the C-17 hydroxylase deficiency form, there is decreased production of both androgens and estrogens and, theoretically, increased production of both DOC and aldosterone. Actually, however, only the DOC levels have been found to be consistently elevated. These patients present with poorly developed secondary sexual characteristics and hypertension. Of course, it is obvious that the diagnosis of Cushing's syndrome and of the adrenogenital syndrome usually is made on other grounds than the suppressed PRA.

Suppressed Plasma Renin Activity in Essential Hypertension

Jerome Conn postulated in 1964 that perhaps 20% of patients who, by all the usual criteria, had essential hypertension actually had "normokalemic primary aldosteronism." This provided the impetus for investigators to measure peripheral PRA in large numbers of patients with hypertension. It now is obvious that at least 20% do, indeed, have the sup-

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Prepared by the Maine Heart Association for this Journal.

PSRO QUESTIONS ANSWERED

At a National PSRO Conference held in New Mexico last February, many interesting and definitive questions came up in the course of the sessions. Among those fielding questions were Dr. Donald Harrington, President of the American Association of Foundations for Medical Care, Senator Wallace Bennett, and Tom Mura of Blue Shield.

Senator Wallace Bennett's speech had two main points of interest:

a. federalization of the Medicaid program and eventual absorption by NHI;
b. that as a "last resort," the Secretary may designate a health insurer as the PSRO in an area where any other organization may be lacking or non-existent.

Following are some of the questions raised during the panel session:

Q. Medical societies will most likely be designated as PSRO's. Won't this result in a medical society locking-in control?

A. No, because the PSRO must be available to all practicing M.D.'s in the area. The societies are the logical place to start a PSRO, and the same society officers can hold office in the PSRO until it is started. Then the Secretary would expect normal election of officers to be held in 3-6 months time.

Q. Will the areas be designated strictly by state lines?

A. No, in some instances, the medical community will be the governing factor even though it may overlap into three states.

Q. Which aspect of the PSRO's will be made public?

A. All data except the patient's individual records. They anticipate some problems with psychiatric records.

Q. Where in HEW will the responsibility for the PSRO's be located?

A. In the office of the Assistant Secretary for Health. There will be a Director and three task forces from SSA, MSA, and one from HSMHA. The total budget is $35 million, $11 million from Medicaid and $24 million from Medicare.

Q. How will PSRO's be reimbursed?

A. On reasonable costs. There are funds available to experiment with different methods such as incentive payments. Mr. Tierney indicated it would be hard to correlate cost and performance and raised the question as to what the PSRO would do with the award money. Would this be incentive or profit?

Q. Because fiscal intermediaries and carriers report to HEW, will there be communication and coordination between HEW and PSRO?

A. Definitely yes.

Q. Presently some states perform on-going review, i.e., hospital utilization review. Who will be responsible for determining whether it is doing a good job, Federal regulations or the PSRO?

A. The PSRO will have that responsibility; if the hospital feels discriminated against, it can appeal. It is required that physicians serving on hospital review committees participate in PSRO.

Q. Will there be interim newsletters and bulletins released from the three task forces?

A. Yes.

Q. Who will evaluate the PSRO's performance?

A. Each state will have a Program Review Team composed of M.D.'s, other professional personnel in the health care field, and consumer representatives (Sect. 229).

Q. Who will review chiropractors, dentists, podiatrists, physical therapists, etc.?

A. The PSRO will maintain a complete patient profile including the care rendered by all of the above medical personnel.

Q. If a designated PSRO fails, what happens to records, etc.?

A. The system will be owned by the government if funds came from the government. If not, only the Medicare and Medicaid records will pass over to the government and to the successor PSRO. If an on-going review organization is designated as the PSRO, the government will pro-rate any funds necessary.

Through the Q and A session, it appeared that money to start and operate PSRO's did not seem to be a problem.

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Department of Health and Welfare

Public Health Aspects of Obesity

HARRY ALLEN, M.D.*

Healthwise we may well say that this is the "Age of Antibiotics." The important infectious diseases of 25 years ago are no longer public health problems. In place of these are the diseases of an aging population, including heart diseases, cardiovascular diseases and cancer. Of particular importance is the relationship of obesity to increased mortality and lowered life expectancy. This is especially true of the members of this group in the 40-50 age bracket. Obesity is directly associated with hypertension and myocardial failure which accounts for a majority of the tragic deaths of men at the apex of their success. In fact, the Framingham long-term study of some 5,000 people showed that obese persons develop cardiovascular diseases about 80% more frequently than persons of normal weight and diabetes about 70% more frequently. After age 45, persons who are 10 lbs. overweight are subject to an 8% increase in death rate; 20 lbs., 18%; 30 lbs., 28%; 50 lbs., 56%. The importance of these statistics is to emphasize the deleterious effects of obesity. More important than statistics is that the majority of cases of obesity are potentially preventable. Obesity is a problem that each of us can work on to increase our own survival and happiness.

Definition:

Obesity is defined as a physical state where excessive fat is stored in the body. It is by no means restricted to men; in fact it is more frequent in women. Overall, it is estimated that one-half of the population or more than 100 million Americans are too fat! It is of little wonder then from this prospective why coronary artery disease is of epidemic proportions in this country.

There are several causes of obesity, among which are disturbances in endocrine gland functions, neurotic overeating, and an inherited propensity for being overweight.

Abnormal functions of the thyroid, hypothalamus, pituitary, adrenal and sex glands all cause obesity. Each of these has specific and characteristic localization of fat deposition. These endocrine disturbances are serious conditions requiring specialized medical management and are not the topic of discussion today. This group makes up only a small percentage of the cases of obesity. The most important and common cause of obesity is a caloric intake persistently exceeding the caloric out-put. In simple language, this means eating too much.

Limitation of energy output (lack of exercise) goes hand-in-glove with overeating. Some of this may be attributed to laziness and some to psychological factors. Especially in the high geared society of today, nervous tensions and frustrations are always present. These may be expressed as "pleasure in good food" and also decrease in physical activity. The psychiatrists suggests that overeating may serve as a substitute when social, business and sexual desires are unsatisfied.

Pathology:

Now the question arises as to just what effect overeating has on the body. It is a known fact there is an increase in vascular circulation resulting in an increased demand on the heart. In addition, there is the constant association of elevated blood pressure commonly called hypertension. Fat is also infiltrated into cardiac muscle, putting further demand on the cardiovascular system. In the same fashion, there are fatty deposits in the walls of the blood vessels, a condition called atherosclerosis. This predisposes to myocardial infarcts or "heart attacks" and cerebrolvascular accident or "strokes." The increased weight in itself results in degenerative arthritis of the joints. There is also increased infection in the skin beneath the fat. Thrombophlebitis and varicose veins are also commonly associated factors.

Treatment:

Early treatment by competent medical practitioners is advised. It is important to get at the underlying cause and establish proper eating habits. The latter is the phase of treatment that we all can participate in. Usually some restriction of diet along with moderate exercise is ordered. Reduction of diet is always necessary. This diet consisting of liberal amounts of protein, would allow for approximately 1100 calories per day for persons having moderate activity or increased to 1500 calories with manual work. Carbohydrates should be supplied in bulky fruits and vegetables. Such a diet allows an ideal weight depending on height and physique.

Actually, the medical profession has been pessimistic concerning any value of exercise in weight reduction. It would take 11 hours of handball, or 7 hours of splitting wood, to take off 1 lb. *However, regular daily exercise of 30 minutes of handball would take off 26 lbs. in one year. We must start young in pursuing sports that can be played through most of life. Among such sports are tennis, golf, swimming, handball, volleyball, cross country*

*Needham, Massachusetts.

skiing, boating, jogging and hiking. This probably would require some reorientation of athletic programs of our schools.

Summary

The problem of obesity is something than can be ameliorated by the complete cooperation of the patient with the medical profession. The happiness of your family necessitates your maintaining optional health. By watching your diet and having regular exercise, you will do much to decrease the waistline. The health department and local doctors stand ready to offer advice as to proper diet and exercise. Also, remember that the best guard to your health is an annual physical examination. Patients with endocrine disorders should seek medical advice as soon as possible.

SUPPRESSED PLASMA RENIN ACTIVITY AND PRIMARY ALDOSTERONISM

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pressed PRA; however, as noted above, most of these do not have elevated or fixed aldosterone levels. Neither do any significant number have Cushing's syndrome or the adrenogenital syndrome. A great deal of effort is being expended at the present time in an attempt to determine why PRA is suppressed in the remainder. A defect in the sympathetic nervous system response to volume depletion has been offered as one explanation. It is more tempting, however, to implicate some unidentified type of mineralocorticoid excess in these patients. In support of the latter, it has been shown that they do have expanded extracellular fluid volumes and that the inhibition of adrenal steroid synthesis by aminoglutethimide or the administration of the mineralocorticoid antagonist spironolactone can result in sodium and water loss and lowering of blood pressure. A search for other possible mineralocorticoids has led to the discovery by Melby and his associates of increased secretion of 18-hydroxy-11-deoxycorticosterone in a few, but certainly not the majority, of these patients.

An important additional factor in attempting to understand the significance of a suppressed PRA is the contention by Laragh and his associates that the finding of a suppressed peripheral PRA has important prognostic implications apart from any possible relationship to etiology. Their data show that patients with hypertension and low peripheral PRA have a more benign course than those with normal or high peripheral PRA and that they are protected from the development of strokes and heart attacks. If this is confirmed by others, it would not only offer another reason for the measurement of PRA in patients with hypertension, but also would imply that the treatment of hypertension should emphasize those antihypertensive drugs, such as alpha methyl dopa and propranolol, that suppress renin secretion.

Recommendation

The efforts to elucidate the significance of a suppressed PRA are still continuing and the answers all are not available. In the meantime, however, a practical guideline is needed as to when the clinician should attempt to demonstrate, with sodium restriction and ambulation, suppressed peripheral PRA. In formulating this guideline, the important point to emphasize is that, at the present time, the measurement of peripheral PRA in a patient who, by all other criteria, has essential hypertension probably will not lead to a cure of the hypertension, nor to a drastic change in therapy. This being the case, it is recommended that, for the present, the attempt to demonstrate suppressed PRA be limited to those patients who have features that suggest the presence of the specific entity, primary aldosteronism. The most important feature, of course, is spontaneous hypokalemia. As implied above, this recommendation may change drastically if further causes of suppressed PRA are defined and/or if the prognostic significance is confirmed.

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Program - 120th Annual Session

Maine Medical Association

June 16, 17, 18, 19, 1973

Shawmut Inn, Kennebunkport

Arranged by the Scientific Committee

ROBERT H. PAWLE, M.D., Falmouth
Chairman

BRADLEY E. BROWNLOW, M.D., Blue Hill

ALAN W. BOONE, M.D., Bangor

The Scientific Program of the annual meeting of the Maine Medical Association is made possible by the cooperation and assistance of the Technical Exhibitors and the following organizations:

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Maine Society of Internal Medicine

Maine Academy of Orthopedic Surgeons

Section on Ophthalmology of the M.M.A.

Neurosurgeons' Group

Maine Psychiatric Association

Maine Radiological Society

Maine Thoracic Society

For this cooperation and support, the members of the Scientific Committee are grateful.

Information

Registration:
Registration throughout the session will be in the Lobby at the Shawmut Inn. Registration fee $2.00.

Saturday, June 16 — 12:00 M. to 5:00 P.M.
Sunday, June 17 — 9:00 A.M. to 5:00 P.M.
Monday, June 18 — 9:00 A.M. to 6:00 P.M.
Tuesday, June 19 — 9:00 A.M. to 4:00 P.M.

Telephone: The number at the Shawmut Inn is Kennebunkport, (207) 967-3931.

Visiting Delegates:
Introduction of Visiting Delegates will take place at meetings of the House of Delegates on Saturday, June 16 and Sunday, June 17.

Technical Exhibits:
This year, twenty-one companies are contributing to the success of the annual session program by participating in the Technical Exhibits. A list

of the exhibiting companies and representatives will be found on page 111 of this program.

Please show your appreciation for the support of these companies by visiting these exhibits.

Badge Code:
Badges with green borders indicate Officers, Past Presidents, Delegates and Alternate Delegates of the M.M.A.; yellow borders, members of the M.M.A.; blue borders, guests; red borders, exhibitors; and plain white for the members of the Woman's Auxiliary.

Saturday, June 16

2:00 P.M. First Meeting of the House of Delegates

Call to Order: GEORGE W. WOOD, III, M.D., President

Presiding: Speaker of the House, GEORGE W. BOSTWICK, M.D.

Presentation of the A. H. Robins' Physician Award for Community Service

Presentation of the Maine Blue Cross and Blue Shield "Award of Appreciation"

7:00 P.M. Dinner

Sunday, June 17

9:30 A.M. Reference Committee Meetings

12:30 P.M. Luncheon

2:00 P.M. Second Meeting of the House of Delegates

Election of President-elect and Executive Committee District Members

General Assembly (Immediately following the House of Delegates)

7:00 P.M. Lobster Bake

Speaker:
MR. ROBERT H. HENRY, Director, Professional Affairs, U. S. Pharmacopeial Convention, Inc., Rockville, Maryland

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Monday, June 18

Scientific Program

9:30 A.M. to 12:30 P.M.

Welcome — BRADLEY E. BROWNLOW, M.D.

9:30 A.M. **Childhood Lead Poisoning**
JOHN W. GRAEF, M.D., Chief, Emergency Services, Children's Hospital Medical Center, Boston, Massachusetts

10:30 A.M. Speaker to be announced

11:30 A.M. **Quality Assurance of Medical Care and PSRO's**
WILLIAM C. FELCH, M.D., President, American Society of Internal Medicine, Rye, New York

12:30 to 2:00 P.M. Luncheon

Scientific Program

1:30 to 4:00 P.M.

Welcome — ALAN W. BOONE, M.D.

Sponsored by the Maine Chapter, American College of Surgeons and the Maine Trauma Committee

1:30 P.M. **Business Meeting, Maine Chapter, American College of Surgeons**

2:00 P.M. Panel Discussion: **The Emergency Room Problem — Everyone's Problem**

Moderator:
DAVID B. PILCHER, M.D., F.A.C.S., University of Vermont College of Medicine, Burlington, Vermont

Panelists:
H. CARL AMREIN, M.D., Madison, Maine

MRS. JEAN ATHERTON, Triage Nurse, Emergency Room, Maine Medical Center, Portland, Maine

WALTER R. PETERLEIN, JR., M.D., Springvale, Maine

DARRELL P. THORPE, M.D., Director, Emergency Division, Maine Medical Center, Portland, Maine

6:00 P.M. Social Hour, Dutch Treat

7:00 P.M. **Annual Banquet**

Presentation of Honorary Pins

President's Address: GEORGE W. WOOD, III, M.D.

Tuesday, June 19

Scientific Program

9:30 A.M. to 12:30 P.M.

Welcome — ROBERT H. PAWLE, M.D.

9:30 A.M. **New Training in Family Practice**
EUGENE S. FARLEY, JR., M.D., Professor and Director, Family Medicine Program, University of Rochester School of Medicine & Highland Hospital, Rochester, New York

10:30 A.M. Panel Discussion: **Fractured Hips Revisited**

Moderator:
WILLIAM R. MACAUSLAND, JR., M.D., Orthopedic Surgeon, Boston, Massachusetts

Panelists:
The Moore Prosthesis
LAWRENCE CRANE, M.D., Orthopedic Surgeon, Portland, Maine

Intertrochanteric Fracture
PAUL STUCKI, M.D., Orthopedic Surgeon, Waterville, Maine

Intracapsular Fracture
ROBERT S. FURMAN, M.D., Orthopedic Surgeon, Rockland, Maine

11:30 A.M. Panel Discussion: **Physicians' Assistants — Should We or Shouldn't We?**

Panelists:
ROBERT G. MACBRIDE, M.D., Lubec, Maine

JOHN P. DOW, M.D., Pittsfield, Maine

DONALD M. ROBERTSON, M.D., Milbridge, Maine

12:30 to 2:00 P.M. Luncheon

Scientific Program

1:30 to 4:00 P.M.

Welcome — ROBERT H. PAWLE, M.D.

Sponsored by the Maine Medico-Legal Society

1:30 P.M. Business Meeting

Presiding — ANTHONY BETTS, M.D., President, Waterville

2:00 P.M. **Narcotic Abuse — The Medical Examiner's View**
MICHAEL M. BADEN, M.D., Deputy Chief Medical Examiner, Office of Chief Medical Examiner, New York, New York; Associate Professor, Department of Forensic Medicine, N.Y. University School of Medicine

6:30 P.M. Dinner

Presentation of Golf Prizes by

DANIEL R. SHIELDS, M.D., Lewiston

Chairman, Golf Tournament

Specialty Group Meetings

Monday, June 18

2:00 P.M. **SECTION ON OPHTHALMOLOGY OF THE M.M.A.**

PAUL E. FLOYD, M.D., Farmington, presiding

Photocoagulation of Fundus Disease
J. WALLACE MCMEEL, M.D., Assistant Clinical Professor of Ophthalmology, Harvard Medical School, Boston, Massachusetts

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2:00 P.M. **MAINE SOCIETY OF INTERNAL MEDICINE**

BENJAMIN ZOLOV, M.D., Portland, presiding

The Future of Internal Medicine
WILLIAM C. FELCH, M.D., President, American Society of Internal Medicine, Rye, New York

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2:00 P.M. **MAINE SOCIETY OF ANESTHESIOLOGY**

PHILIP J. VILLANDRY, M.D., Portland, presiding

4:00 P.M. **NEUROSURGEONS' GROUP**

Business Meeting

DANIEL A. ROCK, M.D., Lewiston, presiding

4:00 P.M. **MAINE THORACIC SOCIETY**

Business Meeting

BRADLEY E. BROWNLOW, M.D., Blue Hill, President, presiding

Tuesday, June 19

11:00 A.M. **MAINE RADIOLOGICAL SOCIETY**

Executive Committee Meeting

HOWARD R. DEMING, M.D., Portland, President, presiding

2:00 P.M. **MAINE RADIOLOGICAL SOCIETY**

Controlling View Box Quality Through Sensitometry
MR. LEONARD F. BOOS, Director, Radiography Training Education Center, Eastman Kodak Company, Rochester, New York

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2:00 P.M. **MAINE CHAPTER, AMERICAN ACADEMY OF PEDIATRICS**

MAURICE ROSS, M.D., Saco, presiding

Public and Private Sectors in the Care of Our Children
MERRITT B. LOW, M.D., District I Chairman,

American Academy of Pediatrics, Greenfield, Massachusetts

2:00 P.M. **MAINE PSYCHIATRIC ASSOCIATION**

PAUL A. JONES, SR., M.D., Union, presiding

The Future of Psychiatry
MAXWELL JONES, M.D., Consultant Psychiatrist, Ft. Logan Mental Health Center, Denver, Colorado

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HONORARY PINS

Presentation of the Association's Honorary Pins will be made by George W. Wood, III, M.D., President of the M.M.A., at the Annual Banquet, Monday evening, June 18 at 7:00 P.M.

FIFTY-YEAR PINS

Fifty-Year Lapel Pins will be presented to the following members who were graduated from Medical School in 1923:

Androscoggin County
Charles F. Branch, M.D., Auburn
University of Vermont College of Medicine

Cumberland County
Abram I. Cohen, M.D., Harrison
Tufts University School of Medicine

Eugene E. O'Donnell, M.D., Portland
Yale University School of Medicine

Theodore M. Stevens, M.D., Portland
Harvard Medical School

Kennebec County
Howard F. Hill, M.D., Waterville
Harvard Medical School

Knox County
Barbara B. Stimson, M.D., Owls Head
Columbia University College of Physicians and Surgeons

Oxford County
Chesley W. Nelson, M.D., Norway
University of Vermont College of Medicine

York County
Herman G. Sandvoss, M.D., Kennebunkport
University of Munich

FIFTY-FIVE-YEAR PINS

Fifty-Five-Year Pins will be presented to the following members who received Fifty-Year Pins in 1968:

Cumberland County
William E. Freeman, M.D., Yarmouth
Bowdoin Medical School

Knox County
Harry A. Naumer, M.D., Port Clyde
Columbia University College of Physicians and Surgeons

Penobscot County
W. Merritt Emerson, M.D., Bangor
University of Vermont College of Medicine

Piscataquis County
Charles N. Stanhope, M.D., Dover-Foxcroft
Bowdoin Medical School

SIXTY-YEAR PINS

Sixty-Year Pins will be presented to the following members who received Fifty-Year Pins in 1963:

Androscoggin County
Ralph A. Goodwin, Sr., M.D., Auburn
Harvard Medical School

Cumberland County
Jacob Melnick, M.D., Portland
College of Physicians and Surgeons, Boston

Albert W. Moulton, Sr., M.D., Portland
Bowdoin Medical School

Kennebec County
Ralph L. Reynolds, M.D., Waterville
Harvard Medical School

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The Connecticut State Medical Society
NORMAN H. GARDNER, M.D., East Hampton
BERNARD O. NEMOITIN, M.D., Stamford

New Hampshire Medical Society
THOMAS G. COGSWELL, M.D., Concord

Medical Society of the State of New York
THOMAS F. MCCARTHY, M.D., Bronx

The Rhode Island Medical Society
JOHN E. FARRELL, Sc.D., Providence

Vermont State Medical Society
HARRY ROWE, M.D., Wells River

Delegates to Out-of-State Meetings

The Massachusetts Medical Society
STANLEY E. HERRICK, JR., M.D., Lewiston

The New Brunswick Medical Society
LINUS J. STITHAM, M.D., Dover-Foxcroft

New Hampshire and Vermont State Medical Societies
DONALD L. ANDERSON, M.D., Lewiston

Medical Society of the State of New York
CHARLES R. GLASSMIRE, M.D., Portland

The Rhode Island Medical Society
LEONARD G. MIRAGLIUOLO, M.D., Bangor

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Thomas F. Shields, M.D.	John W. Carrier, M.D.
Behzad Fakhery, M.D.	Lawrence A. Nadeau, M.D.
Albert Shems, M.D.	Richard A. Marshall, M.D.
Charles W. Steele, M.D.	Richard W. Taylor, M.D.
Gilbert R. Grimes, M.D.	Lionel R. Tardif, M.D.
Aroostook County Medical Society	
Benoit Ouellette, M.D., Secretary	
Eugene G. Gormley, M.D.	Rodrique J. Albert, M.D.
Arthur K. Carton, M.D.	Eric F. Nicholas, M.D.
Cumberland County Medical Society	
Alfred E. Swett, M.D., Secretary	
Stephen E. Monaghan, M.D.	Ronald J. Carroll, M.D.
John R. Davy, M.D.	Harry A. Bliss, M.D.
Irving J. Poliner, M.D.	Paul Maier, M.D.
Peter B. Webber, M.D.	William J. Hall, III, M.D.
Robert G. Sommer, M.D.	Hugh P. Robinson, M.D.
Robert H. Pawle, M.D.	Walter B. Goldfarb, M.D.
Harold N. Burnham, M.D.	Lloyd G. Davies, M.D.
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Douglas R. Hill, M.D.	Henry B. Finks, M.D.
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George I. Geer, Jr., M.D.	Robert P. Timothy, M.D.
Franklin County Medical Society	
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David G. Reed, M.D., Secretary	
Peter E. Giustra, M.D.	Richard J. Kahn, M.D.
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Oxford County Medical Society	
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Thomas Anton, M.D.	Leandre R. Charest, M.D.

Maine Tuberculosis and Health Association
20 Willow Street, Augusta, Maine 04330

Ad Hoc Committee on Contract Inhalation Therapy Service

STATEMENT

At the request of the Board of Directors of the Maine Tuberculosis and Health Association, the president of the Association appointed an ad hoc committee to review and evaluate inhalation therapy services offered to Maine Hospitals on a contract basis by commercial firms. The Ad Hoc Committee was requested to submit a statement to the Board based upon its study of available information and its professional judgement.

Members of the Ad Hoc Committee included representation from Maine Blue Cross-Blue Shield, the Comprehensive Health Planning Agency of Maine, the Maine Chapter American Association of Inhalation Therapists, Maine Hospital Association, Maine's Regional Medical Program and the Maine Thoracic Society. Staff of the Maine Tuberculosis and Health Association assisted the committee.

The Ad Hoc Committee is aware of the Association's interest in and efforts relating to the alarming increase in chronic obstructive lung disease over the past decade. Much of the Association's energies and resources, in cooperation with the Maine Thoracic Society, and other professional groups, have been focused on the education of doctors, nurses and allied health workers regarding the diagnosis and care of patients with chronic respiratory diseases.

As the program evolved, detailed information was sought and obtained regarding the location, availability and extent of RD facilities in hospitals throughout the State of Maine, the most recent activity being the Maine Respiratory Disease Conference of 1971-72. These educational and fact-finding efforts have served admirably to inform physicians and hospital administrators about the need to improve hospital services and facilities for RD patients. The Association has, in part, been responsible for building a favorable climate for the introduction of contract inhalation therapy services into the hospitals of Maine.

The Maine RD Conference Report of 1972 detailed a positive strategy for implementing a state-wide program for improving respiratory care that took full cognizance of economics, advances in diagnosis and therapy of RD and increased sophistication of RD equipment. The program recommended by the RD Conference stresses the regional concept with the implementation of stage I (primary care) and stage II (regional comprehensive center) hospitals and a system of linkage between the community (stage I) and regional (stage II) facilities. The Ad Hoc Committee strongly endorses the regional care concept and urges the Maine TB and Health Association to give high priority towards its implementation.

In addition, the Ad Hoc Committee recommends that the proposed Maine Regional Medical Program demonstration of inhalation therapy services on a shared basis among a group of small hospitals in Hancock County be endorsed by the Maine TB and Health Association. This project could point the way for other groups of smaller hospitals to join forces themselves for the provision of high quality inhalation therapy services.

The Ad Hoc Committee recommends as well that the Association take leadership, in cooperation with other appropriate agencies, in exploring the need for inhalation therapy training within the State of Maine. At the present time, Maine does not have an organized, accredited inhalation therapy training program.

A final positive recommendation offered by the Ad Hoc Committee is that Maine hospitals desiring to implement an organized inhalation therapy service be urged to seek consultation from the Association. The Association, in turn, through resources available to it, should provide expert guidance in assisting health care facilities in developing inhalation therapy services of high quality and appropriateness.

The Ad Hoc Committee does not believe that contract inhalation therapy services will improve respiratory care. Unethical commercial arrangements – the rendering of services without physiologic justification or supervision by qualified medical personnel – are conditions inherent in the contract arrangement.

There is sufficient evidence to suggest that the costs to the patient or third party payers for services provided under contract inhalation therapy services are excessive. The Ad Hoc Committee has outlined positive steps which, if followed, could result in the advancement of the care of respiratory disease patients, consistent with the total development of Maine's health care system, and without adding an escalatory cost burden to the health care consumer.

The Board of Directors recommends that all appropriate measures be taken to advise hospitals, physicians, Associated Hospital Services of Maine, Maine Department of Health and Welfare and other interested agencies and parties of this statement.

March 14, 1973

Committee Members: Omer Breau, Edgar J. Caldwell, M.D., Richard T. Chamberlin, M.D., Gareth O. Jones, M.D., Mark R. Knowles, John LaCasse, Robert Miller, Wilfred A. Poirier, Cora E. Pike, R.N.

Staff: Maine TB and Health Association, George T. Nilson, Mary C. Olsen.

Necrologies

M. Lewis Layton, M.D.

1908-1972

Dr. M. Lewis Layton, 64, of Belfast, Maine died on December 2. He was born in Boston, Massachusetts on May 29, 1908.

Dr. Layton received his medical degree from Middlesex University School of Medicine, Waltham in 1931. He interned at the Ashland Hospital in Pennsylvania and the Whidden Hospital in Everett, Massachusetts and served in the U.S. Army Medical Corp during World War II and the Korean Conflict and held the rank of Lt. Colonel when discharged.

He practiced in Boston for many years and located in Belfast in 1969. Dr. Layton was a member of the Waldo County Medical Society and the Maine Medical Association.

Roswell E. Hubbard, M.D.

1891-1973

Dr. Roswell E. Hubbard, 82, a practicing physician in Waterford, Maine for over fifty years, died on January 19 at his home following a long illness.

He was born in Hatfield, Massachusetts on January 1, 1891, son of Roswell and Fannie Graves Hubbard. A graduate of Smith Academy and Bowdoin College, Dr. Hubbard received his medical degree from Bowdoin Medical School in 1918. He served in the U.S. Navy during World War I, and following his discharge in 1919 came to Waterford and established his medical practice.

Dr. Hubbard was an honorary member of the Oxford County Medical Society and the Maine Medical Association, receiving a 50-year pin in 1968, and would have been eligible for his 55-year pin at the annual session this June. He was also a member of the American Medical Association, a member of the medical staff of the Northern Cumberland Memorial Hospital in Bridgton and the Stephens Memorial Hospital in Norway, and served as Health Officer for the towns of Waterford and Sweden for more than thirty years.

Surviving are his widow, the former Mary Amy Field; two daughters, Mrs. Mary Flynn of St. Petersburg, Florida and Miss Jean Hubbard of Waterford; two sons, Dr. John F. Hubbard of Sterling, Illinois and Roswell E. Hubbard, Jr. of Wayland, Massachusetts; and eleven grandchildren.

Harold E. Libby, M.D.

1910-1973

Dr. Harold E. Libby, 62, of Westbrook, Maine died unexpectedly in a Portland hospital on February 16.

He was born in Chicago on July 23, 1910, son of Earl S. and Ella Michaels Libby. Dr. Libby was graduated from Villanova College, received his medical degree from Temple University School of Medicine in 1936 and interned at the Broadlawns General Hospital in Des Moines, Iowa.

Dr. Libby practiced medicine at Lincoln and Gardiner before coming to Westbrook in 1948 and specialized in the treatment of alcoholism.

He was a member of the Cumberland County Medical Society, the Maine Medical Association, the American Medical Association, the American Medical Society for Alcoholism, the National Group Psychotherapy Association and the Governor's Council for Alcoholism. He was also a member of the Westbrook Community Hospital.

Surviving are his mother and his wife, the former Doreen Howard; a daughter, Mrs. Scott MacStrauich of Richmond, Virginia; a son, Earl C. Libby of Palo Alto, California; and five grandchildren.

Edward A. Greco, Sr., M.D.

1900-1973

Dr. Edward A. Greco, Sr., 72, of Cape Elizabeth, Maine, a prominent Portland cardiologist for many years, died unexpectedly at his home on March 23rd.

He was born in Portland on June 19, 1900, son of Frank and P. Louisa Amborose Greco.

Dr. Greco was graduated from Portland High School, Georgetown University and received his medical degree from Georgetown University School of Medicine in 1928. He interned at Georgetown University and served a residency at the Tuberculosis Hospital in the District of Columbia. He served in the Army during World War I and II. In World War II, he was a Lt. Colonel in the Army 67th General Hospital in Europe.

He began his medical career in Portland's former Maine General Hospital, serving first in the outpatient department and later became senior attending physician until 1946. He established the Cardiology Department at Mercy Hospital and was chief of cardiology from 1946 to 1972, when he was named chief of the Cardiology Department Emeritus.

Dr. Greco was a member and past president of the Cumberland County Medical Association, a member of the Maine Medical Association and the American Medical Association. He was one of the six founders and a past president of the Maine Heart Association; a past vice-president of the New England Cardiovascular Society; a past president of the Maine Thoracic Society and a member of the national organization; a charter member and life member of the American College of Physicians; and chairman for many years of the T.B. Committee of the Maine Medical Association. He was also governor for the Maine Chapter of the American College of Chest Physicians and regent of the New England section of the group for several terms. At the time of his death, Dr. Greco was an honorary regent of the society.

Surviving are his wife, the former Katherine B. Rasmussen; two sons, Dr. Edward A. Greco, Jr. of Cape Elizabeth and Francis P. Greco of Los Angeles; two daughters, Mrs. Joseph H. Salisbury of Cape Elizabeth and Mrs. Paul J. Peters of Vilseck, Germany; a sister, Mrs. Chester Burns of South Portland; eleven grandchildren and two nieces.

John R. Fenger, M.D.

1919-1973

Dr. John R. Fenger, 53, prominent surgeon of Norway, Maine and his wife Jane, died on March 18 in a predawn fire at their home located at Yagger, 6 and one-half miles outside of Norway.

Dr. Fenger was born in Perth Amboy, New Jersey on May 27, 1919, son of John and Marguerite Fenger. He was graduated from Bowdoin College in 1942 and received his medical degree from Yale University School of Medicine in 1945. After an internship at the U.S. Naval Hospital, Dr. Fenger served surgical residencies at the New York Hospital and Memorial Hospital in New York City. He served in the U.S. Navy and attained the rank of Lieutenant Commander.

Dr. Fenger practiced at the Dhahran Medical Center in Saudi Arabia for two years, the Tuxedo Memorial Hospital and Huntington Hospital in New York, and then located in Norway in 1968 where he served as chief of surgery at the Stephens Memorial Hospital.

He was a member of the Oxford County Medical Society, the Maine Medical Association and the American Medical Association; was a fellow of the American College of Surgeons and a past president of the Stephens Memorial Hospital Staff Association.

Surviving are a son, Christopher, who resides in Connecticut; three stepsons, Peter Marshal of San Diego, California, Steven Marshal of Sagertown, Pennsylvania and Chris Marshal.

In Memoriam

Aroostook County	
Gerald H. Donahue, M.D.	Presque Isle
Cumberland County	
Francis M. Dooley, M.D.	Portland
Ralph A. Getchell, M.D.	Cape Elizabeth
Edward A. Greco, Sr., M.D.	Portland
Harold E. Libby, M.D.	Westbrook
Kennebec County	
Aaron Cook, M.D.	Waterville
Rodolphe J. F. Pomerleau, M.D.	Waterville
Oxford County	
John R. Fenger, M.D.	Norway
Roswell E. Hubbard, M.D.	Waterford
Piscataquis County	
Frederick J. Pritham, M.D.	Greenville Junction
Waldo County	
M. Lewis Layton, M.D.	Belfast
York County	
Ansel S. Davis, M.D.	Springvale
Oscar W. Perrault, M.D.	Biddeford
Donald E. Troop, M.D.	Springvale

News, Notes and Announcements

Massachusetts Society of Law and Medicine Annual Dinner Meeting

The annual Dinner Meeting of the Massachusetts Society of Law and Medicine will feature an after-dinner talk by the Honorable Edward F. Hennessey, Associate Justice, Supreme Judicial Court, Commonwealth of Massachusetts on *The Doctor in the Courtroom.*

The meeting will be held at the Marriott Motor Inn, Commonwealth Avenue at Route 128 and Massachusetts Turnpike, Newton, Mass. at 6:30 P.M., Wednesday, May 30, 1973. Nonmembers are welcome.

For further information and reservations, please write or call:

Massachusetts Society of Law & Medicine
454 Brookline Avenue
Boston, Massachusetts 02215
Phone: 617:734-8316

American Board of Family Practice Announces Examination Dates

The American Board of Family Practice announces that it will give its next two-day written certification examination on October 20-21, 1973 in various centers throughout the United States. Information regarding the examination can be obtained by writing:

Nicholas J. Pisacano, M.D., Secretary
American Board of Family Practice, Inc.
University of Kentucky Medical Center
Annex No. 2, Room 229
Lexington, Kentucky 40506

PLEASE NOTE: It is necessary for each physician desiring to take the examination to file a completed application with the Board office. *Deadline for receipt of applications in the Board office is August 1, 1973.*

The Arthritis Foundation Announces Slide Lecture Programs Available

The Arthritis Foundation, Maine Chapter, announces the availability, on loan, to physicians and hospitals, of five slide lecture programs on arthritis.

Titles available include "Differential Diagnosis of Rheumatoid Arthritis," "Rheumatic Diseases In Children and Young Adults," "Clinical Immunology," "The Knee in Rheumatoid Arthritis," and "Clinical Slide Collection on the Rheumatic Diseases."

Each of the programs contains 100 or more 35 mm. slides, most in full color. A comprehensive lecture guide, detailing every slide, is an integral part of the program. A small hand viewer is provided for scanning slides prior to use in a projector or for personal study.

"Differential Diagnosis of Rheumatoid Arthritis" was prepared by George E. Ehrlich, M.D., Temple University Health Sciences Center, Philadelphia. The program correlates the physical features of the various forms of arthritis with roentgenographic and laboratory findings.

"Rheumatic Diseases in Children and Young Adults" was prepared by Mario Spagnuolo, M.D., New York University School of Medicine. The slide presentation is a comprehensive review of juvenile rheumatoid arthritis, rheumatic fever and lupus erythematosus.

"The Knee In Rheumatoid Arthritis," prepared by Alan H. Wilde, M.D. of the Cleveland Clinic Foundation, presents the knee as a model of the disease process in rheumatoid arthritis. Areas covered include mechanisms of joint destruction, clinical problems, radiographic anatomy and surgical treatment.

Arthur J. Amman, M.D. of the University of Wisconsin Medical Center, is responsible for putting together the "clinical Immunology" presentation, which links current thinking to the field of immunology to clinical medical practice.

The collection of "Clinical Slides in the Rheumatic Diseases" places emphasis on the clinical photographs and X-rays in addition to photomicrographs of pathological specimens. Designed by the Visual Aids Subcommittee of the Professional Education Committee of The Arthritis Foundation, the collection contains contributions from approximately 106 physicians.

Physicians, allied health professionals, and hospitals desiring use of these slide learning systems should contact the Maine Chapter of The Arthritis Foundation, Front Street, Post Office Box 333, Bath, Maine 04530, phone 442-8109.

State of Maine Board of Registration of Medicine Secretary — George E. Sullivan, M.D. Waterville, Maine

Physicians Licensed to Practice Medicine and Surgery in the State of Maine

THROUGH EXAMINATION

Ahdieh, Tahereh; Altman, Robert; Amin, Saroja R.; Arain, Abdul M.; Babenco, Jorge; Barcia, Rafael G.; Bassey, Ephraim N.; Batheja, Narain L.; Batri, Antoun; Borkar, Bhagwant B.; Bress, James H.; Cavalieri, John A.; Chang, Hark C.; Coello, Armando J.; Collier, Hugh N.; Corvalan, Jaime G.; Czapla, Andrzej; Deluca, Gerald J.; de Mello, Vincent R.; Deoras, Mukund P.; Deuskar, Sudar S.; Farooki, Mohamed A. M.; Ferrero, Vittorio; Fok, Shiu K.; Frank, Benjamin G.; Gallicano, William J.; Gibson, Stephen K.; Giltman, Larry I.; Ginde, Ravindra V.; Goldstein, Harvey A.; Green, Kenneth W.; Grover, Sukhdev S.; Hariman, Robert J. I.; Hathout, Maher M.; Hermann, George; Hoang, Choon G.; Horvath, Barney C.; Isgut, Alejandro E.; Jonas, Ernesto A.; Kang, Song G.; Khalil, Tawfik A.; Khan, Abdul H.; Kim, Tae H.; Lee, Byung-Joo; Lester, Arthur I.; MacPhail, Jean C.; Majumder, Santi K.; Mallikarjunachary, Vajrakarur; Marignani, Pierluigi; Mathew, Thomas; Mendelovici, Filip; Mujtaba, Qaiyum; Munteanu, Florin D.; Murthy, Kollegal S.; Nair, Sreedharan, D.; Nappi, Anthony; Nudel, Dov B.; Pai, Kochikar G.; Pai, Pundalik P.; Park, Jae-Kil; Parson, Narendra L.; Passidomo, Michael; Patel, Ashok; Pillay, K. Vijayaprasanthan; Plansky, Robert L.; Prasad, Shishir C.; Querijero, Gerardo B.; Quli, Syed H.; Rachelson, Herbert V.; Rao, K. Rama M.; Rahman, Abdul H. M. R.; Salami, Sayed M.; Salgunan, Nambi; Samsamy, Baher; Sarriyanoglou, Paul G.; Scarlata, Robert W.; Schoen, Roy M.; Sekaran, Kamalesh K.; Shahabuddin, Mohammad; Silverman, Beatrice; Steward, Antony M.; Stieglitz, Lewis N.; Strominger, Marius; Susnea, Raftopol, Mihai-Dan; Suzuki, Mitsuaki; Tatpati, Daniel A.; Tatpati, Olga A.; Thakker, Govind; Triner, Lubos; Vasudevan, Ambalapetta K.; Veerappa, Chandiah; Veerappan, Muthuveerappan; Verchese, Kalarikkal T.; Vogel, Barry M.; Vyas, Hansagauri K.; Winter, Peter F.; Yang, Jae Y.; Yu, Ching-Yi.

THROUGH RECIPROCITY

George, Edward A.; Leonidas, Leonardo L.; Stone, Bryan.

County Society Notes

CUMBERLAND

The 371st meeting of the Cumberland County Medical Society was held on October 19, 1972 at the Holiday Inn in Portland, Maine.

Sixty-eight members and guests were in attendance. The usual social hour was omitted and a roast beef dinner served at 6:00 p.m.

Following the dinner, the guest speaker, Mr. Arthur Hughson, City of Portland health officer, was introduced. Mr. Hughson described the organization of the Portland Health Department, discussed its many functions as well as its financial basis. Plans for a regional Public Health Service which will include surrounding towns are already developed and will shortly be implemented.

The business meeting was then called to order. The treasurer's and secretary's reports were accepted as read.

The report of the expenses of the last outing in September was made. It is evident that the attendance at this meeting does not justify this expenditure. A new format will be introduced next year.

First readings of the following applicants were made: Drs. Bruce D. Nelson, Stephen B. Paulding, John Zerner and Paul H. Dumdey. These were referred to the Executive Committee.

The following applications were given second readings and all voted into membership: Drs. David L. Adams, Harry W. Bennert, Jr., Germain A. Binette, Marjorie A. Boyd, Edgar J. Caldwell, Rudy Haak, Stephen M. Soreff and William T. Strauss.

A report from the Medical-Legal Screening Committee was received from Dr. Sylvester. Dr. Charles R. Geer reported for the Peer Review Committee. Dr. Robert E. McAfee then reported on the last meeting of the Maine Medical Association Executive Committee.

Following a lengthy discussion, a motion to increase the Cumberland County Medical Society annual dues to $40.00 per year was introduced, seconded and passed.

A resolution favoring fluoridation of the Portland water supply was presented.

The 372nd meeting of the Cumberland County Medical Society was held on November 16, 1972 at the Stage Coach Inn in South Portland, Maine.

A total of 93 guests and members were present. Our guests included sixteen newly-elected members of the House of Representatives and State Senators. Following a social hour, an excellent dinner was served. Following the dinner, Dr. Douglas R. Hill, President, called the business meeting to order.

Four new members were admitted to membership via transfer from other county societies. These were Drs. John D. Rice, Jr., William C. Taylor, Patrick A. Dowling and Alice H. Parsons. In addition, Drs. Stephen B. Paulding, John Zerner and Bruce D. Nelson were voted into active membership. Dr. Paul H. Dumdey was admitted into Junior membership.

Dr. Hill announced a caucus of the County Delegates to be held December 5, preceding the winter meeting at Bangor. The business meeting was adjourned by Dr. Hill. Following this, our guest speaker, Mr. William Baltaks, New England Field Representative of the American Medical Association, spoke concerning medical legislation.

The major topic of discussion was H.R. 1 which is the most recently passed act concerning the Social Security amendments and Medicare coverage. He reviewed the increased scope of coverage which would be provided and indicated that further study of these would be accomplished under Medicare.

In addition, he discussed the newly passed legislation which now includes optometrist and chiropractic services despite many previous recommendations to the contrary by numerous well-recognized study groups, both in this country and in Canada.

In addition, the Professional Service Review Organization was discussed.

Finally, Mr. Baltaks stressed the importance of the individual physician's local influence, as well as the importance of the Political Action Committee; in particular, the MEMPAC group under the able direction of Dr. Jack Steeves of Skowhegan.

Following this, a lively discussion ensued with much dialogue between members and guests. The following suggestions seemed of importance: First, the presence of a lobbyist representing the Maine Medical Association in Augusta. Second, a service which will provide to the Maine Medical Association a copy of all bills coming before the Legislature. Third, a member of the individual county societies should be assigned as a resource and research person available to each member of the Legislature and State Senate.

Following the discussion, the meeting was adjourned at 9:30 p.m.

The 373rd meeting of the Cumberland County Medical Society was held December 21, 1972 at Valle's Restaurant in Portland, Maine. Following the social hour, an excellent dinner was served.

At the meeting, 53 members and guests were present. The guest speaker, Wilma Jordan, R.N., was introduced by the President, Dr. Douglas R. Hill. The subject of her presentation was of the Portland Visiting Nurse Association. Mrs. Jordan announced that the Portland Visiting Nurse Association has recently combined its services with those of the District and Visiting Nurse Services in Scarborough, Westbrook and Cape Elizabeth. She also discussed the utilization of services and the need for physician referral and participation.

The business meeting was called to order by Dr. Hill. Dr. Wesley J. English discussed plans of the Education Committee of the Cumberland County Unit of the American Cancer Society for free PAP smear clinics to be held in this area sometime next year. Following this, a motion to support the project and request members as physician volunteers was made, seconded and voted.

Dr. Peter B. Webber then presented a resolution concerning the deterioration of the relationship between physicians and third-party insurers. He also noted the increasing numbers of claim forms resulting in more paper work and the resulting increase in office help. The slow response of the third parties and the inability to get adequate answers to many questions affects us all as physicians. Since it seems unlikely, if not impossible, to cope with this problem as individuals, it will best be dealt with on an organized basis. He suggests that a committee be appointed at the county level to receive all communications concerning these problems from the physician members. He further suggests that this committee coordinate its efforts with other groups throughout the county and State, and that we deal with the fiscal intermediaries as an organized group.

Following a lengthy discussion in which many members participated, a motion to appoint such a committee was strongly supported and the President was instructed to appoint members to this Ad Hoc Committee.

Dr. McAfee, our delegate to the American Medical Association, reported to us on the recent meeting in Cincinnati and provided many interesting topics for our discussion and consideration.

One of the major topics concerns the problem of addiction, mental illness, alcoholism and/or other illnesses among physicians. He dealt with the inabilities of society to deal with these problems and suggested a plan of action to be implemented at the county level.

The following applications were then presented for second readings. Drs. Andrew B. Packard and Arthur W. Kaemmer were accepted into membership. First readings in the applications of Drs. Gaylen Johnson, Anthony Salvo and Charles McLeskey were presented and forwarded to the Executive Committee.

Dr. Hill then discussed the recent House of Delegates meeting of the Maine Medical Association in Bangor and noted that further increase in State dues may be upcoming in the future. The reason for this being the increasing need for services at the State level to deal with both legislative and insurance problems.

There being no further business to discuss, the meeting was adjourned at 10:00 p.m.

The 374th meeting of the Cumberland County Medical Society was held on January 18, 1973 at Valle's Restaurant. The wives of members were our invited guests. Approximately 200 members and guests were present. Following a social hour and dinner, a brief business meeting was held.

Dr. Gaylen Johnson was elected to active membership and Drs. Charles McLeskey and Anthony Salvo were voted into junior membership.

Following the business meeting, the guest speaker, Dr. George W. Wood, III of Brewer, President of the Maine Medical Association, was given a rather detailed and hilarious introduction by his good friend and Alternate Delegate to the American Medical Association, Dr. Robert E. McAfee.

Dr. Wood responded in kind and then he presented a discussion of the committee structure of the Maine Medical Association and the activities of the various committees. He stressed the need for more revision of committee structure as well as pointing out the need for increased action by these committees in several crucial areas.

ALFRED E. SWETT, M.D., *Secretary*

KENNEBEC

The Kennebec County Medical Association met at the Silent Woman Restaurant in Waterville, Maine on January 18, 1973. An elegant buffet dinner was served following which the newly-elected President, Dr. Richard R. Dole, presided at the business meeting.

The minutes of the last meeting were accepted as read by the secretary.

The name of Dr. Richard Giuffre from Winthrop was read as a proposed member. His application is to be considered and he will be voted upon at the next meeting of the Association.

Dr. Dole appointed Dr. Richard E. Barron to fill a vacancy on the council for a term of three years. Dr. Dole also appointed Dr. John T. Chen as an alternate delegate for a term of two years. The members who were present were informed of the Interim Meeting of the House of Delegates of the Maine Medical Association to be held on Sunday, April 8, 1973 at 2:00 p.m. No specific Association business was brought up to be presented at this meeting. Dr. Dole then appointed a committee to review the bylaws of the Association. The chairman is Vice-President William E. Schumacher, M.D. Other members of the committee are Drs. Joseph J. Hiebel, James C. Hayes, Richard Barron and Richard T. Chamberlin.

Dr. Richard Chamberlin then reported on the activity of the peer review committee of the County Association over the past year. He indicated that cases have been reviewed almost on a monthly basis. He pointed out that the passage H.R. 1 has established PSRO as the law of the land and that the Maine Medical Association has applied to be the PSRO agency for the State. He expressed the opinion that it would be desirable to maintain the county peer review organization to keep this activity on a local level and he suggested that we have a responsibility to meet the challenge thus presented.

The scientific portion of the program then followed with a most erudite and interesting presentation by Dr. Peter Behan on the topic, "Current Concepts of Neuro-Immunology." His presentation of data and observations derived from his extensive laboratory and clinical research activities indicated the widespread importance of Neuro-Immunology in a broad gamut of neurologic disease.

The meeting was adjourned by Dr. Dole at 9:35 p.m.

KEVIN HILL, M.D., *Secretary*

PENOBSCOT

A meeting of the Penobscot County Medical Society was held on January 16, 1973 at the Tarratine Club in Bangor, Maine, and called to order at 7:40 p.m.

Dr. Benjamin L. Shapero noted the recent illness of Dr. Robert L. Burdick and reported that he would no longer be able to fulfill the duties of Secretary of this Society. Dr. Philip G. Hunter was appointed by the executive committee to fill the secretarial post.

A resolution regarding the position of the Penobscot County Medical Society with regard to chiropractic medicine, as amended, was moved, seconded, and passed. Copies of this resolution are to be sent to all State Senators and Representatives, together with copies to the Presidents of the County Medical Societies throughout the State.

A motion was made, seconded, and passed to include the cost of the monthly dinners which precede the business meeting in the yearly dues of the Society.

A discussion regarding the feasibility of a physician representative acting as an informal lobbyist in Augusta was then brought up before the Society. A discussion revolved around the use of a professional lobbyist in a full-time position or on a part-time basis, as well as a physician or physicians performing a similar function in Augusta.

It was moved, seconded, and passed to invite the Woman's Auxiliary of the Penobscot County Medical Society to join the Society for dinner at the February meeting.

Dr. George W. Wood, III, speaking as President of the Maine Medical Association, addressed the Society on the topic, "What Would You Like Us To Do?" Dr. Wood reviewed and discussed the future roles of the various committees of the Maine Medical Association and where the direction of our efforts should be and what programs we as a Society would like to see put forth. This discussion was most informative and represented a concise analysis of the present status of our committee system within the Maine Medical Association.

Dr. Charles D. McEvoy, Jr. discussed briefly the role of the State "B" agency of the Penobscot Valley Regional Health Planning Council. He requested individuals to consider serving as a member of the agency and expressed his feelings regarding the need for physician participation in this agency role.

The meeting closed with a discussion about the need for a local physician group to whom physicians at the State government level could turn to for information and discussion regarding the health needs of our area. Dr. Shapero stated that he would appoint a three-man committee who would be responsive to the physicians representing State government, and who in turn would act as a spokesman for physicians of our area and our Society.

The monthly meeting of the Penobscot County Medical Society was held on February 20, 1973 at the Pilot's Grill. There were 26 members in attendance.

The topic of the evening's talk was "Avenues of Approach to the Treatment of Alcoholism." The speakers were Dr. Elizabeth Levinson and Mr. Frank Dennis. Mr. Dennis, who is director of the Alcoholic Rehabilitation Center located at the Bangor State Hospital, and who is also associated with the Counseling Center, described briefly his objectives for the treatment of alcoholism as a disease and quite possibly within the confines of the general hospital setting. During the course of his talk and the discussion period which followed, Mr. Dennis provided some pertinent and informative remarks with regards to his feeling about the treatment of alcoholism and his desire to

have physicians within our area utilize the Alcoholic Rehabilitation Center for the treatment of alcoholism.

Dr. Elizabeth Levinson followed Mr. Dennis in providing the Society with her comments in the treatment of alcoholism. She felt that physicians should direct their treatment programs at the basic diagnosis of alcoholism rather than just treating the secondary manifestations, or the admitting diagnosis, of the effects of alcohol. She encouraged physicians to notify the Alcoholic Rehabilitation Center if they do not receive follow-up reports on patients which they refer to this organization. In addition, she encouraged physicians to allow the Alcoholic Rehabilitation Center to be of service to them and their patients. Furthermore, she described the system of data collection which is being used and compiled in an effort to document the effects or results of their program in the rehabilitation of the alcoholic. In addition, this data is meant to identify those areas of the program which are effective and those areas which are ineffective, thereby improving their overall approach to the treatment of alcoholism by directing their efforts into the effective areas.

The monthly business meeting then followed the talks of Dr. Levinson and Mr. Dennis. The Secretary's report was accepted as read. An announcement was made regarding the March meeting which would be devoted to a panel discussion regarding the delivery of health care and the existing and future health systems which may be employed in providing medical care in our area.

Dr. Thomas L. Watt reported to the Society that the committee studying the medical-legal aspects of medicine has and will continue to meet and discuss pertinent aspects of this very important area of discussion. Dr. Watt explained the functions of this committee and how it relates to the legal profession. He asked for continued support and approval of the Society to continue these efforts and proceed toward a more indepth study within this broad atea in order to bring forth recommendations as to the direction we as a Society should take. This permission was granted by a vote of the members present.

Dr. John J. Pearson reported to the Society the present status of the Home Health Care organization, an affiliate of the Counseling Center, and requested discussion regarding the apparently inordinately large fee charged the patient or the third-party payor for their services.

The application for membership to the Society of Dr. William Wilson was presented and voted upon, the secretary then polling those members absent, and was passed unanimously.

Dr. Shapero announced that a pharmaceutical house requested that the Society support a community survey for hypertension and those people found in need of further care be advised to seek medical evaluation by their private physician. This topic was then discussed at some length; a motion was made and seconded that the Society be a part of such a project, but was defeated by a vote of the members present.

As there was no further business, the meeting was adjourned with the next meeting to be held on March 20, 1973 at the Tarratine Club, Bangor, Maine.

PHILIP G. HUNTER, M.D., *Secretary*

LINCOLN-SAGADAHOC

The regular monthly meeting of the Lincoln-Sagadahoc County Medical Society was held at the Ledges in Wiscasset, Maine on January 16, 1973.

The meeting was called to order at 8:40 p.m. by the Vice President, Dr. Peter A. Evans. The minutes of the last meeting were read by the secretary and accepted.

The nominating committee, represented by Dr. Belknap, nominated the following slate of officers:

President: Dr. Peter A. Evans, Brunswick
Vice-President: Dr. Nelson P. Blackburn, Bath
Secretary-Treasurer: Dr. George W. Bostwick, Newcastle
Delegates to the M.M.A. House of Delegates: Drs. Mary J. Tracy, Damariscotta and Anthony J. Horstman, Boothbay Harbor. Alternates: Dr. Elihu York, Brunswick and Frank O. Avantaggio, Jr., Damariscotta
Censors: Drs. John F. Dougherty, Bath, Carl R. Griffin, Jr., Boothbay Harbor and Samuel L. Belknap, Damariscotta
Program: Dr. Louis Bachrach, Brunswick
Diabetes Detection: Dr. David W. Schall, Brunswick

The slate was accepted unanimously, and the secretary was instructed to cast one ballot.

The secretary presented the Treasurer's Report.

Dr. Evans introduced Dr. Hudson, outgoing chairman of the program committee, who introduced Dr. Martin Bell, pediatric surgeon from Portland. Dr. Bell spoke of the medical care of the newborn infant with surgical problems.

The regular monthly meeting of the Lincoln-Sagadahoc County Medical Society was held on February 20, 1973.

The meeting was called to order at 8:36 p.m. by the President, Dr. Peter A. Evans. The minutes of the last monthly meeting were read and accepted as read.

Dr. Evans asked for Committee reports. Dr. Leck reported for the Executive Committee of the M.M.A. mentioning PSRO plans for the State, the situation in Knox County regarding Camden and Pen Bay Medical Center, admissions policies at State hospitals and shortcomings of mental health clinics.

Dr. Bostwick presented the membership application of Dr. Robert M. Hassan, Damariscotta, and the recommendation of the Board of Censors that this application be accepted. The application was unanimously approved by the members assembled.

There being no more business introduced, Dr. Evans turned the meeting over to Dr. Bachrach of the Program Committee. Dr. Bachrach introduced Dr. Charles Morrison, who spoke of the proposed Wiscasset Medical Clinic. His guest speaker, Mr. Robert Ratigan, described the history of the health clinic project. An extremely lively discussion followed. Dr. Dixon moved that this Society go on record as recommending that the Board of Trustees of the Wiscasset Health Center conduct a feasibility study before proceeding any further with plans for a medical clinic. The motion was seconded by many and passed.

The meeting adjourned at 10:40 p.m.

GEORGE W. BOSTWICK, M.D., *Secretary*

ANDROSCOGGIN

The meeting of the Androscoggin County Medical Association was held at Steckino's Restaurant in Lewiston, Maine on March 15, 1973. The meeting was called to order by Vice-President Dr. Gerard L. Morin at 8:00 p.m. with 43 members present.

The minutes of the previous meeting were read and accepted as read.

Application for membership from Dr. Manuel Sangalang was presented under the approval of the Executive Committee of the Androscoggin County Medical Association. It was moved and seconded to accept Dr. Sangalang into membership of the Androscoggin County Medical Association.

The membership was apprised of a letter from Dr. Hanley, Executive Director, Maine Medical Association, dealing with the Title XIX program for Health Screening of Medicaid Children.

Correspondence was read from the Maine Chapter of American Association of Medical Assistants, District II, requesting appointment of another physician to fill the forthcoming vacancy of Dr. Russell A. Morissette. Action will be taken at the next regular meeting.

The officers of the Androscoggin County Medical Association have been invited to the April meeting of the Cumberland County Medical Society. Dr. Raymond Holden, Trustee of AMA, will discuss PSRO, Phase II & III controls, HMO's and Physician's Unions.

A letter from Dr. Charles A. Hannigan to this Association, proposing consideration of legal counsel to represent this membership and individual members of the Association was read.

"The increasing complexities of dealing with third parties and other providers of services require a new look at our man-

agement of these problems. At a recent discussion, it was suggested that legal counsel be acquired by the County Association for these problems. The legal counsel or the law firm would be expected to become fully acquainted with the background, legal information and necessary mechanics in these areas. The individual member of the Association would have access to counsel and, of course, would be expected to pay in relationship to use of counsel, although the County Association would be expected to pay an annual retainer to counsel and would be financially responsible when the problem is one which involves all members of the Association."

This proposal will be further discussed and acted upon at the next regular meeting.

The Finance Committee report was read by Dr. Tibbetts, Sr., and following considerable discussion, the proposal of the committee was tabled to the next regular meeting.

The Secretary read an amended report from Dr. Cyprien L. Martel, Jr. as representative to the Tri-County Health Planning Agency.

Under new business, it was moved and seconded to request a legislation study order regarding Augusta State Hospital and its delivering of psychiatric care to residents of the County and the entire catchment area. This suggestion had been made by Representative Georgette Berube at our January meeting.

The business meeting was then turned over to the program chairman who introduced Dr. George W. Wood, III, President of the Maine Medical Association. Dr. Wood presented an excellent discussion concerning the reorganization of the Maine Medical Association and how the individual members, as well as the County Societies, can participate.

The meeting was adjourned at 9:50 p.m.

RICHARD M. SWENGEL, M.D., *Secretary*

YORK

Dr. Carl E. Richards of Alfred and Sanford was elected President of the York County Medical Society at the annual meeting held at the Officers Club, U.S. Naval Shipyard, Kittery, Maine. Dr. Richards has served as delegate from the County Society to the Maine Medical Association for 31 years and is a Past President of the State organization.

Dr. Irvin Dorfman of Saco was elected Vice-President. Dr. Charles W. Kinghorn of Kittery was elected Secretary-Treasurer for his 44th year in the office. Dr. Melvin Bacon of Sanford was named assistant Secretary-Treasurer.

Approximately 75 doctors, their wives and guests attended the affair, which opened with a social hour and dinner. The principal speaker was Dr. Dean Siebert, assistant dean of regional medical affairs and associate professor of community medicine at Dartmouth Medical School, Hanover, New Hampshire. He spoke on "Present Day Trends in Continuing Medical Education," illustrating his talk with slides and T.V. presentations.

The Society and the Auxiliary held separate business meetings.

Dr. Ruth E. Endicott of Ogunquit, outgoing President, conducted the meeting of the Medical Society.

Matters relative to the State and county medical organizations were discussed by Dr. Richards and by Dr. Robert F. Ficker of Kennebunkport, York County member of the Maine Medical Association's Executive Committee.

Dr. Robert Vigue of Sanford, who is engaged in the practice of ophthalmology, was voted into membership.

Committees were named for the coming year. Serving on the executive committee will be the officers, plus Dr. Robert S. LaFond and Dr. Walter R. Peterlein, Jr.

Delegates to the Maine Medical Association are Dr. Richards, Dr. Badi M. Haq and Dr. Thomas Anton. Alternates are Dr. Kenneth E. Leigh, Dr. Maurice Ross and Dr. Leandre R. Charest.

The censor committee is composed of Dr. Marion K. Moulton, Dr. Paul S. Hill, Jr. and Dr. Alvin A. Hoffman.

Serving on the peer review committee are Dr. Hill, Dr. Anton, Dr. Bacon and Dr. Leigh.

Dr. Leigh, Dr. Bacon and Dr. Charles S. Turville were named to serve as the nominating committee for 1974.

CHARLES W. KINGHORN, M.D., *Secretary*

WANTED

Issues of the Maine Pharmaceutical Association *Proceedings*, 1867-1905. Desperately needed by the Portland Public Library, 619 Congress Street, Portland, Maine 04101. Original copies were unfortunately lost by the bindery. Have historical value tracing development of pharmacy in Maine.

Contact: Mrs. Barbara Smith, Assistant Director, Portland Public Library. 207-773-4761.

Letters to the Editor

To the Editor:

Re article "A Team Approach to Renal Dialysis in a Community Hospital," Feb. 1973, pages 38, 39

Due to neglect on my part, we failed to mention that the Hemodialysis Unit is at Seton Hospital, Waterville, Maine and that any inquiries regarding the Hemodialysis Program should be addressed to Joseph J. Hiebel, M.D. or Mrs. Penny Bresnick, R.N. at Seton Hospital, Chase Avenue, Waterville 04901.

JOSEPH J. HIEBEL, M.D.
Chief of Medicine

To the Editor:

With the advent of the Modern Voluntary Army and the end of the draft a shortage of military physicians will no doubt exist in the near future. In order to conserve and best utilize military medical officers, the Armed Forces will cease to use these doctors at the AFEES (Armed Forces Examining and Entrance Station).

There is now an effort being put forth to recruit civilian physicians for full time duty at these stations. Retired medical officers are of course in an ideal position to step into such a job by virtue of their past experiences with the military. Others are equally welcome to inquire about further details and are encouraged to do so. The Chief Medical Officer at an AFEES will be given a GS12 status and earn in the neighborhood of $20,000 per year. Provisions have been made to allow for dual compensation of these physicians (i.e., retirees may continue to draw their pensions while functioning as AFEES medical officers).

A complete job description, interview, and information concerning application procedures may be obtained by contacting either Major Thomas P. Maliska, Commander, or Captain Kenneth G. Kraemer, Medical Officer, at the Armed Forces Examining and Entrance Station, 151 Forest Avenue, Portland, Maine 04101 or by telephoning 775-3131, Extension 526 or 523.

KENNETH G. KRAEMER
CPT, MC
Medical Officer

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of the
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Lead Screening Program in Maine

SUSAN J. RINGER*

The Bureau of Health, Maine Department of Health and Welfare, is currently conducting a lead poisoning screening program to identify preschool children in Maine who have abnormal blood lead levels. Habitual ingestion of lead, if undetected, may cause brain damage, blindness, kidney disorder, and possibly death.

Lead intoxication among children has been the subject of recent medical and social investigation. Most studies to date have dealt with the urban dweller, primarily those of the lower socio-economic strata. Very little information is available about lead intoxication among rural children. The only study to identify the prevalence of lead intoxication in Maine was done in Portland during July-August, 1970. The period prevalence rates of this study were similar to those quoted in several studies in large metropolitan areas (approximately 4%). From this study one could ask: If a city of 100,000 has the same lead poisoning problem as does a city of several million, can we expect the prevalence to be similar in a town of several thousand? Results of the current survey should provide the answer.

Another objective of the Lead Screening Program is to increase physician awareness and index of suspicion for lead intoxication. The signs and symptoms of lead intoxication are non-specific and easily mistaken for many childhood illnesses. A child, often between the ages of 1 and 3 years, with episodic vomiting, loss of appetite, some lethargy or hyper-irritability, vague lack of progression of learned skills and mild anemia may have lead intoxication, especially in the summertime. Frequently, a history of pica is absent. Obviously, other diseases can produce similar symptoms, but unless lead intoxication is discovered, the child may eventually cease to ingest lead, the symptoms diminish, and the child pronounced well. The undetected lead intoxication may have caused residual brain damage. The relationship between minimal lead intoxication and residual cerebral dysfunction is not clear, but enough evidence for such a relationship exists to make early diagnosis and treatment essential.

Below is a brief discussion of the clinical aspects of lead intoxication to serve as a guide to increased awareness of the problem and how to handle it.

To produce the disease, lead must enter the body, usually in quantities over 1 mg. per day. The oral and inhalation routes are the usual means of entry. Oral ingestion of lead (other than from food) is most commonly attributed to lead paint. In spite of federal controls, some interior paints contain sufficient lead to cause disease. Most exterior paints are still lead based. Plaster and caulking compounds around window panes have lead in them. Even if the interior of a house has been recently painted, the old layers of paint underneath may contain lead. Lead water pipes (outlawed in current installations) are another source of intoxication.

Inhalation is also a dangerous route. Burning old battery casings for heat in the Blue Ridge Mountains of Virginia is a common source of intoxication. Some authorities have blamed tetra-ethyl lead auto exhaust fumes for the high base line or "normal" blood lead values in cities.

The signs and symptoms of lead intoxication are: anorexia, irritability, lethargy, vomiting (usually forceful and episodic), ataxia often preceded by subtle changes in behavior and loss of motor skills, anemia (the blood picture is similar to iron deficiency anemia), seizures and coma. Any one individual may have some or all symptoms. They may come and go depending on exposure, or the spectrum of signs and symptoms may progress rapidly to death if large doses are ingested over a short period of time.

*Director, Childhood Lead Screening Program, State of Maine, Department of Health and Welfare, Augusta, Maine 04330.

The diagnosis rests on demonstrating the presence of lead in the body in sufficient quantities to produce the signs and symptoms. There is still a controversy over the best test method to determine the amount of lead in the body: hair, urine or blood analysis. Most recent studies show blood testing to be most specific and sensitive. Further controversy is involved between macro and micro-method blood lead sampling. Micromethods are still relatively new, but greatly facilitate mass screening procedures. The method being used for Maine's Lead Screening Program is Anodic Stripping Voltammetry. Capillary blood samples are obtained from a child's finger, ear lobe or heel in lead-free capillary tubes and analyzed at the State Public Health Laboratory. Investigation of the method and evaluation of test results showed Anodic Stripping to be satisfactorily reliable and most suitable at this time for the testing program in Maine. Other methods will be investigated as they are developed.

Interpretation of blood lead values vary according to different authors. The following values have been adopted for Maine's program:

* Less than 40 micrograms per 100ml % indicates non-intoxication.
* Between 40ug % and 60ug % indicates exposure and in the absence of symptoms should be retested within one month to determine if it is rising or dropping.
* Between 40ug % and 60ug % with symptoms and positive ancillary tests should be treated.
* Over 60ug % indicates intoxication and should be treated appropriately.

Ancillary tests to confirm lead poisoning are:

* Blood count, looking for iron deficiency picture.
* Abdominal x-rays (flat plate), looking for radio-opaque flecks in the intestines.
* Wrist and knee x-rays, looking for lead lines.

Several studies have demonstrated a seasonal variation in blood lead (lower in winter, higher in summer) in both the intoxicated and control groups; so in winter, borderline values should be interpreted more stringently.

Specific therapy is aimed at chelating the lead ion and removing it from body tissue. Treatment guidelines are as follows:

Children with blood lead over 60ug % with evidence of acute encephalopathy (lethargy and stupor or coma, seizures, and evidence of increased intracranial pressure) should be admitted to the hospital and treated with 1 dose intramuscularly of BAL (2,3 dimercaptopropanol) 4mg/kg, then 4 hours later begin a regimen of BAL (2.5mg/kg/dose) and CaEDTA (edathamil calcium disodium — 12.5mg/kg/dose) given IM every 4 hours for 5-7 days. Supportive treatment of the increased intracranial pressure and fluid and electrolyte disturbance are necessary.

For children with blood lead over 60ug % with no evidence of acute encephalopathy, outpatient care has been successful. EDTA is given daily for 5 days using 50mg/kg injected intramuscularly (use 1 ml of 1% procaine in the same syringe to decrease pain at injection site). A blood lead is taken the day treatment is begun, then again 1 week after the 5th day of the course. The 5-day course is repeated until the blood lead level drops to 60ug %. Blood is tested again 4 weeks after EDTA therapy is completed. If prolonged therapy is necessary, then it should be given for 3 months then stopped for 1 month, then restarted, etc.

For children with blood lead levels between 40ug % and 60ug % on the repeat sample following initial screening, EDTA may be given as above, but these cases should be individualized.

Treatment is always a poor substitute for prevention. It is imperative to remove the source of lead from the child. Environmental investigations will be conducted in the homes of all positive reactors to identify the lead sources and assist in removal. Parents must supervise their children closely to prevent ingestion of lead chips, and all loose paint in the home must be scraped off and disposed of. Toys and furniture should also be checked for lead paint.

Lead intoxication is most common among low income groups, but obviously all children are potentially susceptible. Physicians diagnosing or strongly considering lead poisoning are urged to report the cases to the Lead Poisoning Screening Program (289-2727), and, if necessary, assistance in determining the source of the lead will be arranged. Assistance in diagnosis and treatment procedure can also be provided.

Postscript

This article was written in November 1972. To date, the Lead Screening Program has tested over 5,000 children, ages 1-6, and has identified a Statewide prevalence of 8%. On the basis of results obtained from the initial survey, plans for an on-going Lead Poisoning Control Program are being developed. A complete report of the Maine Lead Screening Program covering the period July 1972 to June 1973 will be available in the near future.

Acknowledgment

The technical assistance of Dr. Timothy Townsend in the preparation of this article, and the financial assistance from the Division of Child Health are gratefully acknowledged.

References

Sachs, H. K., et al: "Ambulatory Treatment of Lead Poisoning: Report of 1155 Cases," Pediatrics, September 1970, Volume 46, Number 3.

Kotok, D.: "Development of Children with Elevated Blood Lead Levels, A Controlled Study," Journal of Pediatrics, January 1972, Volume 80, Number 1.

Chisolm, J. J.: "Screening Techniques for Undue Lead Exposure in Children, Biological and Practical Considerations," Journal of Pediatrics, November 1971, Volume 79, Number 5.

Barnett, H. L.: Pediatrics, 14th edition, Appleton-Century-Crofts 1968.

Trigeminal Neuropathy Heralding Scleroderma

A Report of a Case

PAUL D. THOMPSON, B.S.* and GEORGE J. ROBERTSON, M.D.**

Sensory loss in the distribution of the trigeminal nerve associated with scleroderma is a recently reported clinical entity. Beighton *et al*[1] reported the first case in 1968. Since then, four more cases have been published.[2,3] In addition, Kaltreider *et al*[4] cite a personal communication from H. Kaiser at the National Institute of Health who has seen three cases of trigeminal neuropathy in scleroderma. The case presented here is believed to be the ninth case in the literature of trigeminal neuropathy in scleroderma. It is remarkable in being the third reported case of such loss preceding the more classic signs of scleroderma.

CASE REPORT

A 48-year-old white female was admitted to the New England Medical Center Hospital on 10/16/72 complaining of right facial numbness and thickening of the skin on the dorsum of her hands. A year prior to admission while brushing her teeth before breakfast, she noted that the right side of her mouth and face was numb, as if she had received novocaine. Eleven months prior to admission she noted stiffness of her knees and ankles that waxed and waned. Ten months prior to admission she was evaluated at another hospital where skull series, brain scan, E.E.G. and pneumoencephalogram were normal. Three months prior to admission she noted tightening of the skin on the dorsum of her hands. She denied pain associated with the facial numbness. She also denied Raynaud's phenomenon, dyspnea, and G.I. complaints.

Physical exam showed a middle-aged female in no distress. Blood pressure was 120/80. The hands had thickened, hyperpigmented skin on the dorsum limiting flexion of the fingers. Neurological exam showed decreased sensation of the trigeminal nerve's second and third division of the right, and third division on the left. Routine lab studies including BUN and creatinine were normal. Rheumatoid factor and anti-nuclear antibody tests were negative. A two-hour postprandial blood glucose was 105 mg %. A protein electrophoresis was normal. Urine was negative for uroporphyrin, coproporphyrins, and heavy metals. Pulmonary function tests were within normal range. Esophageal motility studies showed absence of peristalsis in the esophagus and borderline low resting pressure in the lower esophageal sphincter which did not relax on swallowing. Deltoid muscle biopsy showed increased collagen with no myositis. Skin biopsy was consistent with scleroderma. The patient was discharged on range of motion exercises and symptomatic treatment for esophageal reflux.

Gordon and Silverstein[5] recently reviewed the neurological findings in one hundred and thirty cases of scleroderma. Twenty-four (18.5%) of these cases developed neurological symptoms. In four cases, the cause was felt to be iatrogenic. Eight cases had neurological findings secondary to scleroderma involving organs other than those of the CNS. These findings included uremia and vascular accidents related to renal disease and resultant hypertension. Eight cases were considered "incidental," and four cases had myopathies. The authors postulated that the paucity of direct CNS involvement in scleroderma was related to the fact that the CNS is poor in connective tissue and that cerebral arteries differ from other arteries in lacking an external elastic lamina and in having little media. They expected more peripheral nerve involvement than they found because peripheral nerves are rich in connective tissue.

Christopher and Robinson[6] have found such peripheral involvement with rigorous testing. They studied conduction velocity in three motor nerves and two sensory nerves in patients with scleroderma and found conduction velocity decreased in one of the motor and both of the sensory nerves. They felt this result was due to the greater susceptibility of sensory nerves to injury.

The trigeminal neuropathy of scleroderma is specifically sensory in nature. No motor loss has been reported. Pain is conspicuously absent in these patients. In contrast, Spillane and Wells[7] studied sixteen cases of isolated trigeminal neuropathy from other causes and found pain to be a frequent feature. Other collagen disease including Sjörgren's Syndrome, systemic lupus erythematosus, and dermatomyositis have been associated with trigeminal neuropathy.[2,3]

In scleroderma, more classic signs of the disease have preceded the trigeminal neuropathy in all cases except for the present case and two others.[1,3] Casey and Lawton[3] reported a fascinating case where a sixteen-year-old girl developed Raynaud's phenomenon of the tongue and trigeminal neuropathy some 18 months before skin changes and classic Raynaud's phenomenon appeared. In the cases presented by Ashworth and Tait,[2] however, Raynaud's phenomenon of the hands always preceded the trigeminal involvement. The trigeminal neuropathy, once developed, is permanent. The development of the neuropathy is always sudden requiring days to a few weeks. Our patient's disturbance seemed to develop overnight.

The speed of development raises the question of etiology. Entrapment of the trigeminal nerve in connective tissue has been assumed to be the cause. Rich-

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ter[8] reported a case of sensory loss in all four extremities in a patient with skin hyperpigmentation. No antemortem diagnosis was made, but at autopsy the spinal peripheral nerves were incarcerated in sclerosing fibrous tissue. It is possible that the trigeminal nerve divisions are similarly incarcerated somewhere along that route either by the epineurium and perineurium or some other connective tissue. The speed with which the deficit develops casts doubt on this mechanism. Vasculitis, on the other hand, is well known in collagen disease and can cause such rapid progression. Kaltreider and Tabal[4] reported trigeminal nerve involvement in Sjörgren's Syndrome which they felt was related to vasculitis. Since vasculitis is more frequent in collagen diseases other than scleroderma and since the collagen diseases tend to blend into one another, trigeminal neuropathy in scleroderma might presage the development of other collagen diseases. At present, there is no indication that this is true. Also, there is presently no indication that trigeminal neuropathy in scleroderma is a poor prognostic sign.[2] Since heart, lung, or kidney involvement are the most serious prognostic signs in scleroderma, and these were absent in our case, the prognosis is probably good.

It is impossible to diagnose collagen disease preceded by isolated trigeminal neuropathy. Other causes of neuropathy should be ruled out. A careful search for other signs of collagen disease such as joint pain, skin hyperpigmentation and tightening, and Raynaud's phenomenon should be done to guard against that situation where "a single symptom may obtrude itself so prominently that such signs of primary disease when present may be overlooked."[8]

References

1. Beighton, P., Gumpel, J. M., and Cornes, N. G. M.: Prodromal Trigeminal Neuropathy in Progressive Systemic Sclerosis. Annals of Rheumatic Diseases 27: 367, 1968.
2. Ashworth, B., and Tait, G. B. W.: Trigeminal Neuropathy in Connective Tissue Disease. Neurology 21: 609, 1971.
3. Casey, E. B., and Lawton, N. F.: Progressive Systemic Sclerosis Presenting with Raynaud's Phenomenon in the Tongue and Sensory Trigeminal Neuropathy. Rheumatology and Physical Medicine 11: 131, 1971.
4. Kaltreider, H. B., and Tatal, N.: The Neuropathy of Sjörgen's Syndrome: Trigeminal Nerve Involvement. Annals of Internal Medicine 70: 751, 1969.
5. Gordon, R. M., and Silverstein, A.: Neurologic Manifestations in Progressive Systemic Sclerosis. Archives of Neurology (Chicago) 22: 126, 1970.
6. Christopher, R. P., and Robinson, H.: Studies of Nerve Conduction in Patients with Scleroderma. Southern Medical Journal 65: 668, 1972.
7. Spillane, J. D., and Wells, C. E. C.: Isolated Trigeminal Neuropathy: A Report of Sixteen Cases. Brain 82: 391, 1959.
8. Richter, R. B.: Peripheral Neuropathy and Connective Disease. Journal of Neuropathology and Experimental Neurology 13: 168, 1954.

BULLETIN

ATTENTION DOCTORS

We need qualified physicians to do consultative examinations in specialty fields. Examinations are scheduled in your own office. Reimbursement approximates your charge to general public. If you are interested, write to:

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Audit Section From Principles of Practice*

Charles S. Burger, M.D., John C. Bjorn, M.D. and Harold D. Cross, M.D.**

Our Principles of Practice is a set of rules or statement of philosophy defining our approach to major areas of our practice such as audit, use of paramedical personnel, patient responsibility, etc. The development of a practical and just system of audit is a problem that is going to be of concern to all physicians in the coming years as a result of H.R. 1 and rising consumer demand.

In consultation with Dr. Lawrence Weed, we have given considerable thought to this subject. The following is the section from our Principles of Practice which defines our approach to audit at this point in time. We offer it here in hopes that it may provide food for thought for others struggling with this difficult subject and that critical feedback will help us sharpen our own thinking.

Audit

Dr. Lawrence Weed has stated the basic requirements necessary for rational medical audit! He has further directly contributed to this section of the Principles of Practice.

Physician activity (or that of any other medical personnel) is part of a system of medical care. Audit of this activity requires that: first, the system must be described; secondly, the users of the system must be audited for performance within the rules of the system; thirdly, the system itself must be audited and updated through analysis of the data it generates — feedback from outcomes. Failure to make a distinction between the system itself and poor performance within the system may lead to confusion and injustice in the audit process.

It is recognized that an unstructured audit without rules and criteria can degenerate into no audit at all. At least three approaches to audit have been described:

1. AUDIT STRUCTURE BASED ON ARBITRARY CRITERIA FOR SINGLE PROBLEMS, applied and interpreted in the absence of, and unintegrated into a defined and structured universe of data appropriate to the welfare of the whole individual.

While it may appear desirable to develop protocols and algorithms for the diagnosis, evaluation and management of common problems, it is unlikely that these can be developed in the foreseeable future for more than a fraction of the total problems seen. Also, without computerization it is impossible to require strict application of these. Additionally, patients having problems for which no protocol is available and patients whose total problem list presents unique management problems due to interrelated problems and/or drug interactions defying protocol, deserve assurance that their problems are being adequately managed.

There are difficulties in establishing arbitrary criteria for most problems even when viewed in isolation. To develop criteria for all problems and to enlarge each of these to include the interactions with all other associated problems consistent with the personal goals of a given patient, applicable in all environments and resource situations, is manifestly impossible at present.

2. OUTCOME ANALYSIS: Based on the outcome of a patient's disease without consideration and evaluation of the process of care.

When performance of individuals within the system of care is judged on the basis of outcome of patients with specific diseases and without consideration of the context created by a complete problems list, it is very likely that injustices will result. (See section on "Audit of the System")

3. BEHAVIORAL ANALYSIS: Evaluation of the medical personnel by the behavioral traits of thoroughness, reliability, analytic sense and efficiency as they operate within the context of a defined system of care.

We have chosen the latter as the means of auditing individual performance within the system of care. We believe it is more fair in the face of current realities. Furthermore, it will accommodate the non-memory based approach to logic pathways inherent in the computerized Problem Oriented Medical Record (POMR) when that becomes available. Outcome analysis, we believe, is a more appropriate tool for auditing the system itself and is discussed in this context in a later section. (See "Audit of the System")

The course of each patient through an almost infinite number of biological and social variables is unique. Therefore, the auditor can only judge the logic of each step in the context in which it was taken at the time. The components that go to make up this context can be defined at the outset. Just as the components of a chess game can be defined at the outset, even if the infinite pathways through it cannot be.

The structure of the POMR and the rules for its use facilitate such an audit. The burden is on the auditor to demonstrate from the literature and/or from basic physiological and biochemical principles that any step in medical action is unwise in a given context. (In the not too distant future the pathways through the displays that form the basis for the com-

*Revised 1/73.

**PROMIS CLINIC, Hampden Highlands, Maine 04445.

puterized POMR can be an integral part of the literature available to the physician as he works.)

Description of the System (For the PROMIS Clinic)

The organizing principle of the system is problem orientation of data, and the principal tool in the system is the Problem Oriented Medical Record (POMR).[2,3] The POMR requires:

1. A DEFINED DATA BASE (history, physical examination and laboratory data) that is age and sex specific designed to reveal medical, social, psychiatric and demographic problems.

   A physician must know the universe of facts within which he is to be judged and from which he is expected to formulate the problems. The size and nature of the data base must be appropriate to the type of population he sees, the size of his load and the availability of resources. In those instances in which the agreed upon data base for comprehensive care or the agreed upon minidata base for episodic care has not been obtained, the first problem on the problem list should be Incomplete Data Base due to: (and state reason).
2. A COMPLETE LIST OF PROBLEMS including social, psychiatric, environmental, and medical problems formulated at the highest level of synthesis the physician understands and for which there is sufficient evidence in the data base.

   "Sufficient evidence" will be considered acceptable if the physician under questioning or by reference can point to support in a recent text book of medicine, not older than 5 years, or a recent journal article from one of the standard journals. A low level of synthesis will be acceptable if the plan for each problem is well conceived and appropriate to carry the problem to a higher level of synthesis. In other words, the absolute level at which a doctor states a problem is not as important as the logic and persistence he demonstrates in the pursuit of the problem to either a conclusion or a necessary steady state.
3. NUMBERED AND TITLED PLANS appropriate for each specific problem with subsections for:
   a) Dx (diagnostically) — getting more information to delineate the problem further and more information to manage it. ("Rule outs" are stated here)
   b) Rx (treatment) — what is to be done.
   c) Pt. Ed. (patient/family education) — what they are told.
   d) Goal statement if not implicit in Dx, Rx and Pt. Ed.

   "Appropriate" will be anything which can be supported by a recent textbook or article and which is logical within the context of the other problems of the patient and consistent with the goals of the patient himself.
4. NUMBERED AND TITLED PROGRESS NOTES with subsections for subjective and objective data, assessment and plans appropriate to the problem.

   "Appropriate" will be conscientious pursuit of the original plan or logical modification explained by new data or new knowledge requiring reinterpretation of old data and plans.

Audit of Individuals Performing Within the System

Once the Problem Oriented system described has been established, audit of individuals within the system (in the four phases of medical action, i.e., a defined data base, a complete list of problems, numbered and titled plans and numbered and titled progress notes) should be based on the behavioral traits of thoroughness, reliability, sound analytic sense and efficiency. (See "Rules for the Auditor")[4]

Efforts to date at the PROMIS Clinic in Hampden Highlands, Maine for the audit of physician and paramedical performance for thoroughness, reliability and efficiency have been described.[5] Portions of the audit at this level (almost exclusively in the data base section) can be performed by persons with a minimum of medical experience.

In regard to analytic sense, what is expected of the medical personnel is stated in the description of the system above. Audit at the analytic level deals directly with uncommon as well as common problems at the outset and emphasizes the development of a core of physician behavior which will lead him to more effectively function as a "guidance system" problem solver no matter what the situation. This, in the last analysis, is the patient's greatest guarantee of quality. A physician will pass or fail by the logic of his definition and pursuit of problems within the rules of the POMR.

As stated above, the logic and completeness of his approaches will be considered adequate if he can support them with standard textbooks or standard journal articles not more than 5 years old. This will emphasize his development as a problem solver, using references and principles wisely and emphasize his own need for postgraduate self-education.

The computerized POMR will greatly facilitate his behavior as a problem solver; the very tool a doctor or nurse uses to do his work has built into it the parameters of guidance and the current information needed to do the work correctly. Poor performance should be followed by measures that make personnel looks things up or read carefully what is before them on the computer.

Audit of the System

One might question the choice of a 5-year interval

for "current" medical information. A physician might, for example, be able to justify the use of oral hypoglycemic agents in treating uncomplicated, obese, maturity onset diabetes with an article 3 or 4 years old. More recent literature may not substantiate his choice of treatment. The physician in this instance "passes" and the system "fails." This may occasionally be a problem with our approach to audit but we do not expect it to happen with sufficient frequency to interfere with the operation.

The ultimate test of the system design will be outcome as observed on the patients in our practice. The outcome of medical and social problems separately and in the aggregate can never be attributed wholly to the process of care, recorded or unrecorded. Outcome is dependent upon all variables, known and unknown; process of care deals consciously only with some of the known variables. To the extent that outcome can be correlated reliably with some of the known variables, it should be used to readjust the process of care to give maximally good yields. Therefore, outcome studies should be used to intelligently modify the system of care employed, that is, modification of the content of the data base and modification of the literature (including computer displays) from which problem statements and plans are logically generated. Dr. Stuart Graves has discussed what it means to use outcomes to modify the system.[6,7]

Audit of the system may reveal that the main failure is neither in design, nor in performance of individual personnel, but rather in resources, or leadership resulting in failure to discipline and educate personnel in an effective manner. Each organization must establish its own disciplinary measures and means of enforcement. Outside auditors must see that such measures exist and are employed when indicated. Otherwise, all else may be in vain in some situations.

Implementation

Physician audit of thoroughness, reliability and analytic sense already occurs in an informal, inefficient fashion because of the natural flow of patients from physician to physician in the office. Also, hospitalized patient records are reviewed each time that the physician on call for the week changes.

Audit of paramedic performance at their level of responsibility is already ongoing and has been described in "Audit in a Problem Oriented Practice"[5] and "The Role and Training of Paramedics at PROMIS Clinic."[8,9]

Audit of physician performance in the formulation of problems, plans and follow-up will be conducted on random samples of patient records and based on thoroughness, reliability and analytic sense. Examples of the methodology by which this can be carried out have been described on an appended audit sheet, which outlines the pertinent questions used to evaluate each behavioral trait as applied to each phase of the medical action.

This will begin in January 1973. In order to gain familiarity with the new system physicians within the practice will audit each others' comprehensive examinations done on patients within the last month using the appended audit sheet which defines the pertinent questions. Three charts per week will be done for the first several months. Physicians will be graded on a pass-fail basis in each phase of medical action for each behavioral trait.

At the end of six months, the frequency of audit and means of tabulation will be more clearly defined. Internal audit will continue until improvement in physician performance has plateaued. At that time, plans for the introduction of audit by physicians outside the practice will be formulated.

It is anticipated that in a three-man group where each physician has demonstrated his dedication to the POMR and the audit principle formal disciplinary actions will not be necessary; it is hoped the prime motivation will be pride and conscience.

It is our belief that with this system of audit the individuals' needs for postgraduate education are automatically defined and the caliber of this education will be continuously evaluated.

It is anticipated that audit will be extended to all areas of function relative to patient care in the future (i.e., financial, receptionist, laboratory).

Audit of Physician Performance
PROMIS CLINIC

I. Data Base:
   A. Thoroughness:
      1. Patient Profile: Reason for CPX, description of usual day, family structure/social setting.
      2. Present Illness/Questionnaire:
         a. No audit on narrative questionnaire* positives. Rules for accountability for positives on new questionnaire* have been defined. (1/73)
         b. Chief complaints listed should have present illness or be an attribute.
         c. Is there subjective/objective section for each problem?
      3. Physical Examination: All parts of defined P.E. should be done or reason why not stated (e.g. no sigmoidoscopy — patient refusal).
      4. Lab/physiologic testing: All defined as part of comprehensive data base to be done or reason why not stated.
   B. Reliability: Periodic audit of paramedic exams. None on M.D.'s at present.
   C. Analytic Ability:
      1. Present Illness: Should be sufficient data to support the level of the problem statement.
   D. Efficiency: Defined by office schedule (4-6 CPX/day + follow-ups and acutes).

II. Problems List:
   A. Thoroughness:
      1. Are all agreed upon physical/physiologic/lab abnormalities listed as either problems or as attributes of another problem?
      2. No accountability on ROS positives until new questionnaire.* (1/73)
      3. "Incomplete data base" listed where appropriate?
   B. Reliability: Is problems list up to date — new problems added, resolved noted?

C. Analytic: Are problems defined at a level consistent with the data?
D. Efficiency: None.

III. Plans:
A. Thoroughness:
1. Are there plans for each problem?
2. Are there sections for getting more information (Dx), therapy (Rx) and Patient Education? Need not be where Plan is "none." Also need not list all three for each problem but information recorded should be listed under proper section.
3. R/O's — are they complete and explicit?
4. Management parameters: What parameters are to be followed at what frequency on chronic or life threatening situations? May refer to flow sheet or protocol when these exist.
5. Therapy: Explicit (dose, frequency)? Meds listed on medication chart? Would patient understand the Rx plan?
B. Reliability: If problem stated at a low level of resolution and no R/O's listed, is there evidence that some help is being sought (consult, check textbook, etc)?
C. Analytic Ability:
1. Are goals clear? When defined by protocol are reasons for accepting less than defined goal noted (e.g. in hypertension side effects from meds or azotemia may limit)?
2. R/O's: Are these reasonable in context, logical and supportable? Should be a statement indicating return visit.
3. Therapy: Is this reasonable in context, logical and supportable? Should be a statement indicating return visit.
D. Efficiency: See Follow-up.

IV. Follow-up:
A. Thoroughness:
1. Progress Notes:
a. Are they problem oriented?
b. SOAP format?
c. Plans: Sections for Dx, Rx, Pt. Ed.?
d. New problems added and described.
2. Flow sheets:
a. All appropriate parameters followed?
B. Reliability:
1. Progress Notes:
a. Evidence that initial plan carried out?
2. Flow Sheets:
a. Are they kept up to date?
C. Analytic:
1. Progress Notes:
a. Assessment logical?
b. Plans — as above?
c. Data with appropriate problem?
d. Active problems followed with reasonable frequency?
D. Efficiency:

References

1. Weed, L. L.: Quality Control and the Medical Record, Archives Internal Med. 127: 101-105, 1971.
2. Weed, L. L.: Medical Records, Medical Education and Patient Care, Year Book Med. Publishers, 35 East Wacker Drive, Chicago, Illinois. 1969.
3. Bjorn, J. D. and Cross, H. D.: Problem-Oriented Practice, Modern Hospital Press, Chicago, Ill. 1970.
4. Weed, L. L.: Rules for the Auditor, in The Problem Oriented System, Eds: Hurst and Walker, Medcom Press, 2 Hammarskjold Plaza, N.Y., N.Y. p. 105-110, 1972.
5. Graves, Stuart: Better Records — First Step to Better Quality. Mod. Hospital April 1971, p. 105-108.
6. Graves, Stuart: Clinical Investigation. In Problem-Oriented System, Vol. II. Ed. Hurst and Walker, Medcom Press, N.Y., N.Y. 1973.
7. Burger, Charles: Audit in a Problem-Oriented Practice. In Problem-Oriented System, Vol. II. Ed. Hurst and Walker, Medcom Press, N.Y., N.Y. 1973.
8. Hodges, Eileen: The Role and Training of Paramedics at PROMIS Clinic. In Problem-Oriented System, Vol. II. Ed. Hurst and Walker, Medcom Press, N.Y., N.Y. 1973.
9. Bjorn, John: Physician Assistant — Second Level Entrepreneur? Journal Maine Medical Association 62: 133-138, 1971. (Also reproduced in The Problem-Oriented System, 1972).

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Foreign Body Ingestion With Perforation of One of the Great Vessels of the Mediastinum

Report of a Case and Review of the Literature

ROBERT BOKAT, M.D., JAMES FIFE, M.D. and ROBERT GALEN, M.D.

The ingestion of foreign objects by children is not an uncommon event. However, the report of penetration of one of the great vessels of the neck in association with ingestion of a sharp foreign body is a rare situation.[1]

CASE PRESENTATION

An 11-month-old Negro infant presented to his primary physician on 3/11/70 with a complaint of fever, fussiness and a small amount of vomiting. A urine culture was done at that time because the infant had been operated upon in January 1970, for a tight meatus and posterior uretheral valves, but this culture proved to be negative. The only finding on 3/11/70 was rhinitis. On that visit, Actifed® was prescribed. He continued to be febrile and fussy and was started on Ampicillin on 3/13, which didn't alter his clinical course. On 3/15, following breakfast, which he took poorly, he vomited a quantity of clear fluid and then "a large quantity of blood," for which he was hospitalized at the Regional Memorial Hospital, Brunswick, Maine.

PHYSICAL EXAMINATION: Revealed a lethargic, but alert, Negro infant, who was well-hydrated and well-nourished. There was dry blood on both hands and some mucoid sanguinous material on his palate. The respiratory rate was 40 and cardiac rate 110 and regular. The chest was clear. The abdomen was soft with good bowel sounds. The liver was palpated 1 cm. below the right costal margin.

LABORATORY DATA: On admission, his HCT was 25% falling to 19% on 3/19. HMG 7gms. %, WBC 11,100, Retic count 0.9%. No sickle cells seen. Differential: Segs 76%, Bands 7%, Lymphs 10%, mono's 7%, Platelets 290,000. Smear of RBC's revealed moderate to marked hypochromasia with some bizarre forms. Urinalysis was normal except for a trace of albumin. On 3/17, WBC was 18,350. Bilirubin: Direct negative; Indirect 0.1%.

HOSPITAL COURSE: The child continued to run a temperature of 102°-103°. On his second hospital day, he developed a tachycardia with cardiac rate 150-208. His ampicillin was continued.

On the afternoon of 3/17, he passed a large, black, tarry stool. An upper G.I. series was performed on 3/18. Initial films revealed a large open safety pin in his stomach (Fig. 1). Films of chest at the same time showed a superior mediastinal mass on the left which was interpreted as "thymus versus paraesophageal abscess and mediastinitis." There was no free air beneath the diaphragms. The patient was given 155 cc. of blood which raised the HCT to 25% and a gastrostomy was carried out on 3/18; the safety pin removed. The patient's temperature fell to normal and he did well until 3/22, when his fever reached 104° and persisted. Repeat chest x-rays with barium per gastric tube (Fig. 2), showed the left mediastinal mass, but no free air in the mediastinum. On 3/24, the patient was transferred to the Maine Medical Center. At that time, his temperature was 104.6°, his pulse was 180, and respirations were 72. He appeared quite ill on arrival at the Maine Medical Center with hot skin, moderate dyspnea, lethargy and pale nail beds. HCT on admission was 24%, WBC 23,850 with 85 segs and 2 bands. At 9:35 p.m., 3/24, while inserting a cut down, the patient had a cardiac arrest and was resuscitated. In spite of 250 cc. of whole blood, a left chest tube which produced 100 cc. of red fluid, and medical measures, the patient succumbed at 1:45 a.m., 3/25/70. In retrospect, no history of ingestion of the safety pin could be obtained.

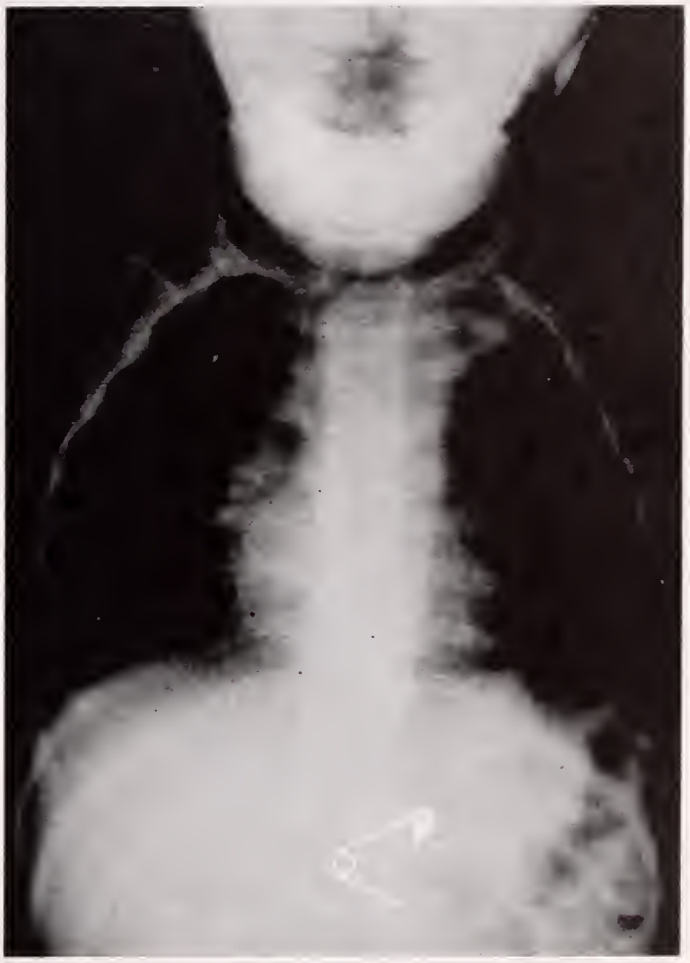

Fig. 1

PERTINENT AUTOPSY FINDINGS: Autopsy revealed perforation of the esophagus on the left side, with a pseudoaneurysm of the left subclavian artery contiguous with the perforated area of the esophagus.

DISCUSSION

Fistulous communications between the esophagus and the heart, pericardium or the other great vessels of the mediastinum are a rare occurrence. Laubscher[2]

Fig. 2

reported a case of communication between the esophagus and left atrium in a 20-year-old male who had "trauma to the esophagus as a child" without further elaboration about the lesion. In 1957, McDaniel and Knepper[3] reported a case of esophageal pericardial fistula and reviewed 19 earlier reports of such fistulae. They noted that 5 cases were due to erosion of the pericardial sac by an ulcer, 5 were produced by introduction of a foreign body into the esophagus and 5 others were associated with carcinoma. Ulcerations of a nectrotic lymph node or congenital diverticulum and trauma related to repair of a lower esophageal diverticulum were attributed to the rest.

Henry and Miscall reported the occurrence of an aortic-esophageal fistula in a four year old, mentally retarded girl, following the ingestion of a coiled spring. Barrie and Townrow,[4] in a review of the literature in 1946, reported 80 cases of aortic-esophageal fistula. They stressed the following consistent features of such a condition, which apparently are applicable to any esophageal-great vessel perforation, which were demonstrated clinically by our patient: (1) The perforation is usually caused by a sharp object which has been ingested. (2) The aorta, presumably due to its proximity to the esophagus is the great vessel most commonly involved. (3) Following ingestion of the sharp object, chest pain occurred in the majority of cases, receded for a variable time, but generally returned before death from hemorrhage. (4) The most striking similarity in such conditions, and which our case illustrated, was a consistency with which a signal hemorrhage preceded the fatal hemorrhage. The interval between signal hemorrhage and fatal hemorrhage in Henry and Miscall's case was a period of 7 days. In Barrie and Townrow's series, the period between signal hemorrhage and fatal hemorrhage averaged 1 day and ranged from 2 hours to 3 days. Our patient had survived 10 days from his signal hemorrhage to his fatal hemorrhage and the latter event might have been precipitated by his struggling during the cut down.

Spasm of the arterial wall, hypotension from hemorrhage and occlusion of the fistula by clots have all been speculated upon as the pathogenesis of this latent period. Miscall and Henry suggested the following sequence in the pathogenesis of the latent period. The esophagus is perforated by a sharp object at or near the level of the aortic arch with leakage into the mediastinum. The sharp object may either injure the wall of the great vessel in proximity with the esophagus at that point or pass through it. The object then may pass through the alimentary tract spontaneously, or as in our patient may have to be removed surgically. Sepsis follows in the wall of the esophagus and great vessel. A fistula then evolves from infection, perhaps abetted by digestion of the esophageal contents. The initial bleed may then exert a tamponade effect or force the formation of a pseudoaneurysm which then recurs as a fatal and massive hemorrhage with progress of infection and digestion. There is no report, to our knowledge, in the literature of a successful repair of an esophageal-great vessel fistula. Even though it is a rare occurrence, this type of accident could, therefore, be considered uniformly fatal.

On the other hand, the prevention of an open "safety pin" (inappropriately named in this case) is a simple task in preventive medicine for the doctor. And even if such an event occurs, the consistency with which a signal hemorrhage takes place — the more alert the physician should be to the possibility of esophageal-great vessel perforation and with consequently immediate attention to the thoracic area.

It would seem prudent early in the well-care of any child to add the admonition, "Close the safety pin!"

Summary

An infant who accidentally ingested an open safety pin which perforated a great vessel in the mediastinum is presented. Implications of such an incident are discussed with particular reference to the sign of a "signal bleed" in mediastinum great vessel perforation. Preventive aspects of the situation are stressed.

Acknowledgment

We would like to extend acknowledgment to Mrs. Evangeline Gault, who so kindly typed the manuscript.

References

1. Henry, Walter J.; Miscall, Laurence: Aortic-esophageal fistula. J. Thoracic Cardiovascular Surgery 39: 258-262, 1960.
2. Laubscher, Frederick A.: Esophagocardiac fistula, report of a case. N.E.J.M. 14: 794-795, 1971.
3. McDaniel, J. R.; Knepper, P. A.: Esophago-pericardial, fistula: report of a case and review of the literature. J. Thoracic Surgery 34: 173-176, 1957.
4. Barrie, H. J., and Townrow, V.: Perforation of the aorta by a foreign body in the esophagus, report and review of the literature. J. Laryngol. 61: 38-42, 1946.

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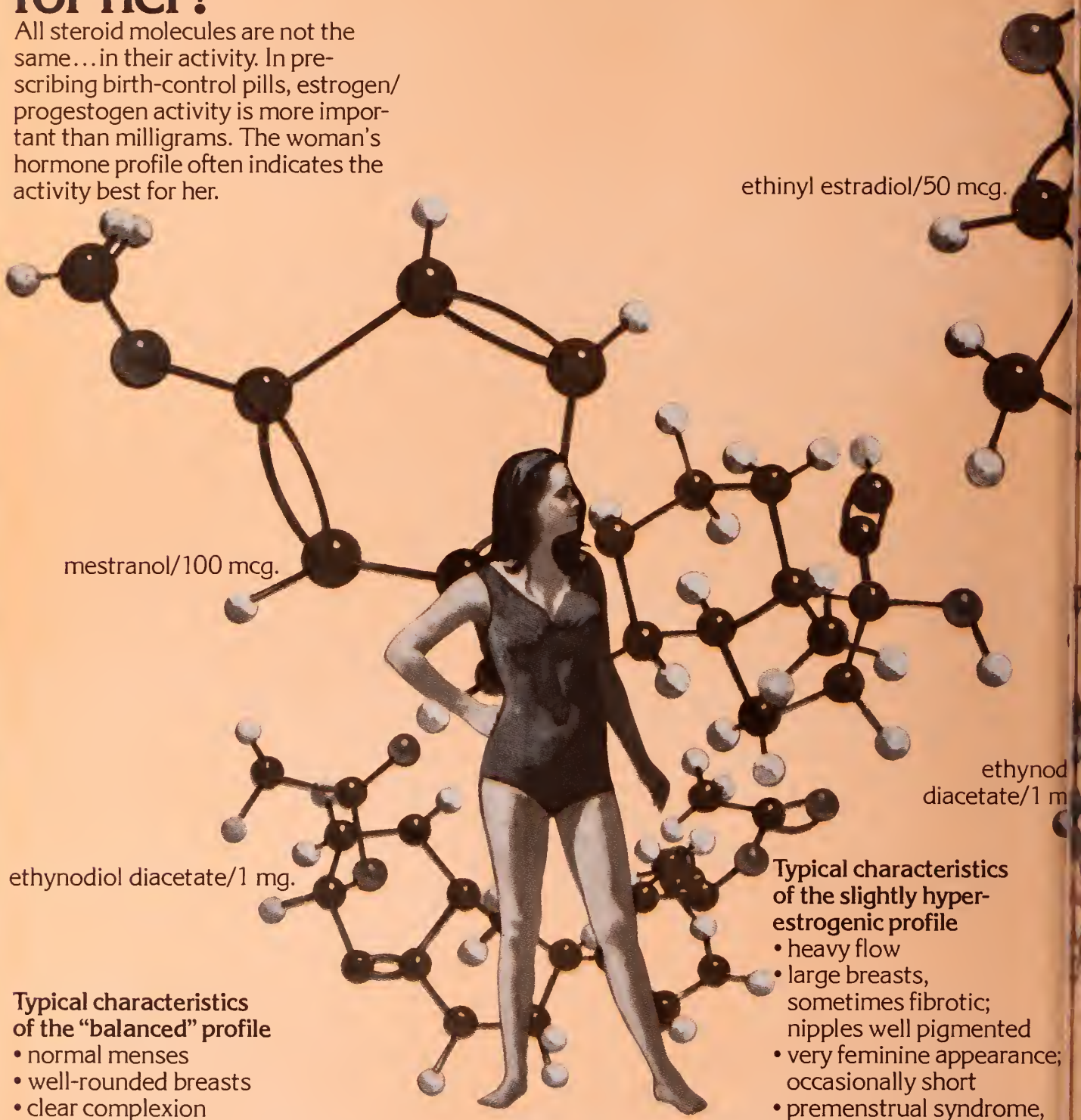

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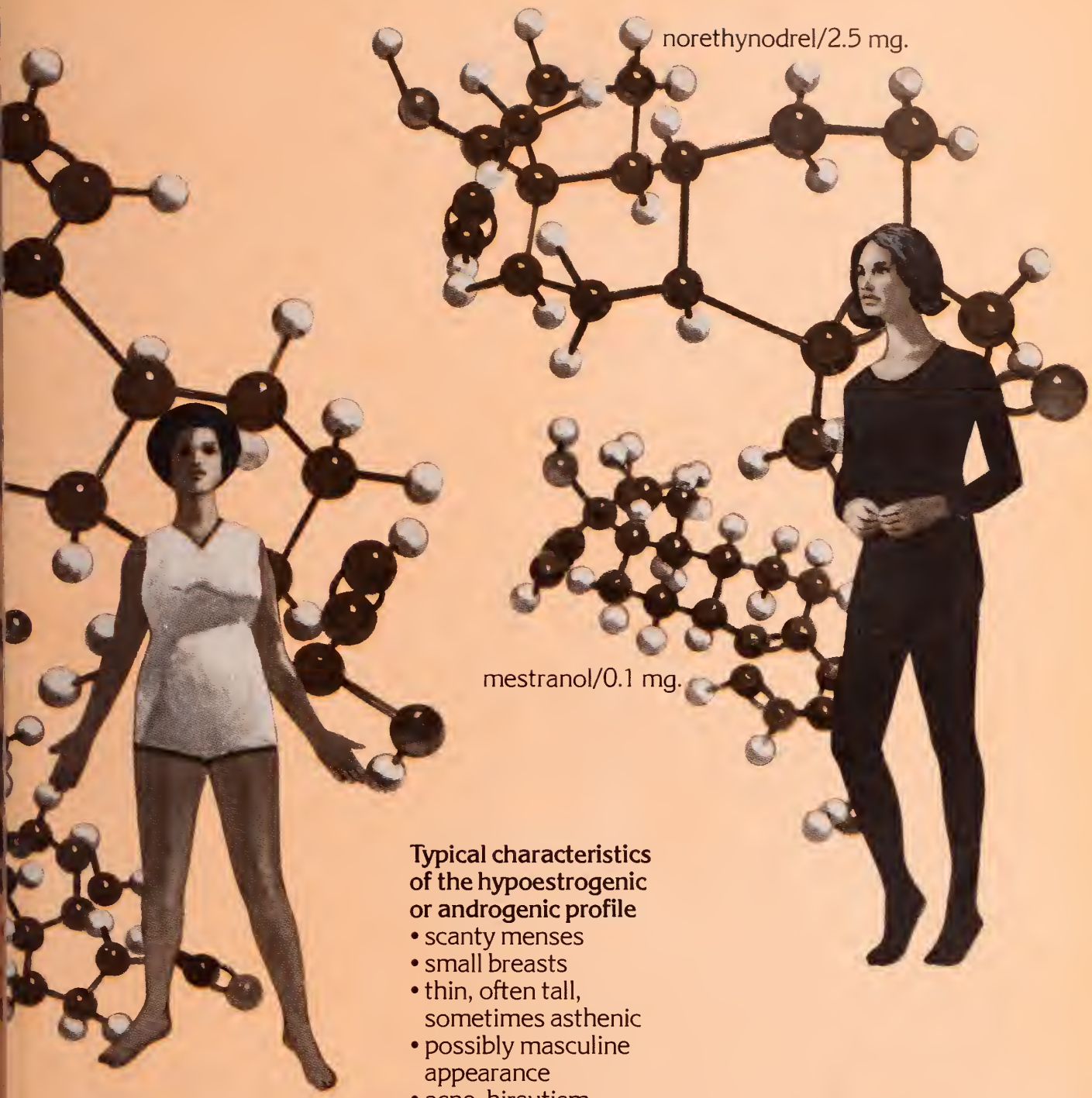

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Actions—Ovulen and Demulen act to prevent ovulation by inhibiting the output of gonadotropins from the pituitary gland. Ovulen and Demulen depress the output of both the follicle-stimulating hormone (FSH) and the luteinizing hormone (LH).

Special note—Oral contraceptives have been marketed in the United States since 1960. Reported pregnancy rates vary from product to product. The effectiveness of the sequential products appears to be somewhat lower than that of the combination products. Both types provide almost completely effective contraception.

An increased risk of thromboembolic disease associated with the use of hormonal contraceptives has now been shown in studies conducted in both Great Britain and the United States. Other risks, such as those of elevated blood pressure, liver disease and reduced tolerance to carbohydrates, have not been quantitated with precision.

Long-term administration of both natural and synthetic estrogens in subprimate animal species in multiples of the human dose increases the frequency of some animal carcinomas. These data cannot be transposed directly to man. The possible carcinogenicity due to the estrogens can be neither affirmed nor refuted at this time. Close clinical surveillance of all women taking oral contraceptives must be continued.

Indication—Ovulen and Demulen are indicated for oral contraception.

Contraindications—Patients with thrombophlebitis, thromboembolic disorders, cerebral apoplexy or a past history of these conditions, markedly impaired liver function, known or suspected carcinoma of the breast, known or suspected estrogen-dependent neoplasia and undiagnosed abnormal genital bleeding.

Warnings—The physician should be alert to the earliest manifestations of thrombotic disorders (thrombophlebitis, cerebrovascular disorders, pulmonary embolism and retinal thrombosis). Should any of these occur or be suspected the drug should be discontinued immediately.

Retrospective studies of morbidity and mortality conducted in Great Britain and studies of morbidity in the United States have shown a statistically significant association between thrombophlebitis, pulmonary embolism, and cerebral thrombosis and embolism and the use of oral contraceptives. There have been three principal studies in Britain[1-3] leading to this conclusion, and one[4] in this country. The estimate of the relative risk of thromboembolism in the study by Vessey and Doll[3] was about sevenfold, while Sartwell and associates[4] in the United States found a relative risk of 4.4, meaning that the users are several times as likely to undergo thromboembolic disease without evident cause as nonusers. The American study also indicated that the risk did not persist after discontinuation of administration and that it was not enhanced by long-continued administration. The American study was not designed to evaluate a difference between products. However, the study suggested that there might be an increased risk of thromboembolic disease in users of sequential products. This risk cannot be quantitated, and further studies to confirm this finding are desirable.

Discontinue medication pending examination if there is sudden partial or complete loss of vision, or if there is a sudden onset of proptosis, diplopia or migraine. If examination reveals papilledema or retinal vascular lesions medication should be withdrawn.

Since the safety of Ovulen and Demulen in pregnancy has not been demonstrated, it is recommended that for any patient who has missed two consecutive periods pregnancy should be ruled out before continuing the contraceptive regimen. If the patient has not adhered to the prescribed schedule the possibility of pregnancy should be considered at the time of the first missed period.

A small fraction of the hormonal agents in oral contraceptives has been identified in the milk of mothers receiving these drugs. The long-range effect to the nursing infant cannot be determined at this time.

Precautions—The pretreatment and periodic physical examinations should include special reference to the breasts and pelvic organs, including a Papanicolaou smear since estrogens have been known to produce tumors, some of them malignant, in five species of subprimate animals. Endocrine and possibly liver function tests may be affected by treatment with Ovulen or Demulen. Therefore, if such tests are abnormal in a patient taking Ovulen or Demulen, it is recommended that they be repeated after the drug has been withdrawn for two months. Under the influence of progestogen-estrogen preparations pre-existing uterine fibromyomas may increase in size. Because these agents may cause some degree of fluid retention, conditions which might be influenced by this factor, such as epilepsy, migraine, asthma, cardiac or renal dysfunction, require careful observation. In breakthrough bleeding, and in all cases of irregular bleeding per vaginam, nonfunctional causes should be borne in mind. In undiagnosed bleeding per vaginam adequate diagnostic measures are indicated. Patients with a history of psychic depression should be carefully observed and the drug discontinued if the depression recurs to a serious degree. Any possible influence of prolonged Ovulen or Demulen therapy on pituitary, ovarian, adrenal, hepatic or uterine function awaits further study. A decrease in glucose tolerance has been observed in a significant percentage of patients on oral contraceptives. The mechanism of this decrease is obscure. For this reason, diabetic patients should be carefully observed while receiving Ovulen or Demulen therapy. The age of the patient constitutes no absolute limiting factor, although treatment with Ovulen or Demulen may mask the onset of the climacteric. The pathologist should be advised of Ovulen or Demulen therapy when relevant specimens are submitted. Susceptible women may experience an increase in blood pressure following administration of contraceptive steroids.

Adverse reactions observed in patients receiving oral contraceptives—A statistically significant association has been demonstrated between use of oral contraceptives and the following serious adverse reactions: thrombophlebitis, pulmonary embolism and cerebral thrombosis.

Although available evidence is suggestive of an association, such a relationship has been neither confirmed nor refuted for the following serious adverse reactions: neuro-ocular lesions, e.g., retinal thrombosis and optic neuritis.

The following adverse reactions are known to occur in patients receiving oral contraceptives: nausea, vomiting, gastrointestinal symptoms (such as abdominal cramps and bloating), breakthrough bleeding, spotting, change in menstrual flow, amenorrhea during and after treatment, edema, chloasma or melasma, breast changes (tenderness, enlargement and secretion), change in weight (increase or decrease), changes in cervical erosion and cervical secretions, suppression of lactation when given immediately post partum, cholestatic jaundice, migraine, rash (allergic), rise in blood pressure in susceptible individuals and mental depression.

Although the following adverse reactions have been reported in users of oral contraceptives, an association has been neither confirmed nor refuted: anovulation post treatment, premenstrual-like syndrome, changes in libido, changes in appetite, cystitis-like syndrome, headache, nervousness, dizziness, fatigue, backache, hirsutism, loss of scalp hair, erythema multiforme, erythema nodosum, hemorrhagic eruption and itching.

The following laboratory results may be altered by the use of oral contraceptives: hepatic function: increased sulfobromophthalein retention and other tests; coagulation tests: increase in prothrombin, Factors VII, VIII, IX and X; thyroid function: increase in PBI and butanol extractable protein bound iodine, and decrease in T^3 uptake values; metyrapone test and pregnanediol determination.

References: 1. Royal College of General Practitioners: Oral Contraception and Thrombo-Embolic Disease, J. Coll. Gen. Pract. 13:267-279 (May) 1967. **2.** Inman, W. H. W., and Vessey, M. P.: Investigation of Deaths from Pulmonary, Coronary, and Cerebral Thrombosis and Embolism in Women of Child-Bearing Age, Brit. Med. J. 2:193-199 (April 27) 1968. **3.** Vessey, M. P., and Doll, R.: Investigation of Relation Between Use of Oral Contraceptives and Thromboembolic Disease. A Further Report, Brit. Med. J. 2:651-657 (June 14) 1969. **4.** Sartwell, P. E.; Masi, A. T.; Arthes, F. G.; Greene, G. R., and Smith, H. E.: Thromboembolism and Oral Contraceptives: An Epidemiologic Case-Control Study, Amer. J. Epidem. 90:365-380 (Nov.) 1969.

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The *Special Note*, *Contraindications*, *Warnings*, *Precautions* and *Adverse Reactions* listed above for Ovulen and Demulen are applicable to Enovid-E and should be observed when prescribing Enovid-E.

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Editorial

Cancer of the Lung – Forty Years Later

Just forty years ago, on April 5, 1933, Dr. Evarts Graham first successfully removed an entire lung for cancer, thus introducing a modality of treatment that has been performed thousands of times during the past forty years. The purpose of this editorial is to describe what has happened in the management of this disease over the last forty years and what trends are evident, especially in the State of Maine.

We are all aware that bronchogenic carcinoma has been the number one killer of males from malignant disease for sometime, and it has also become increasingly evident in the last two or three years, that the incidence in females is increasing markedly. In spite of the excellent editorial, "Thoughts on Lung Cancer in Maine," by H. Richard Hornberger, M.D. in this Journal in March 1969, the late inoperable and unresectable cases, by far outnumber the early cases coming to the thoracic surgeon. This is even more evident among the female patients in my own experience.

Since the first pneumonectomy forty years ago, the techniques of operation have improved considerably. We have learned to do lobectomies whenever possible, rather than sacrificing a whole lung. The preoperative and especially the postoperative care is much more sophisticated. There are many qualified thoracic surgeons. In spite of these technical advances, the overall cure rate in various series remains at 5% or 6%.

A cursory review of the Cancer Registry here at the Mercy Hospital of Portland through 1971, reveals a total of 244 cases of cancer of the lung and of these, 20 (8%) are living over two years. It is interesting that of the two who are 16 years post-pneumonectomy, one had multiple glandular metastases and the other was felt to have transected tumor on the pathological specimen. Another case was explored and biopsied and felt to be unresectable and is living 5 years after a palliative course of cobalt, while one that was biopsied by mediastinoscopy, and felt to be inoperable is living two years after a palliative course of cobalt.

What can we, as physicians, do to improve our results in bronchogenic carcinoma? In the first place, cultivate a high index of suspicion, realizing that men under forty seldom have CA of the lung and that it is quite common in men over fifty who smoke. They are the patients who should have routine, yearly chest x-rays. All undiagnosed shadows on x-rays should be given a work-up to eliminate the possibility of cancer. Anyone in that age group with upper respiratory symptoms should have a complete diagnostic work-up. We should realize that all of these same criteria are now increasingly applicable to women who smoke. Thus far, although chemotherapy has not had much to offer, and tumor immunology appears to have great promise, excision of lung, lobes, or segments and radiation or combinations of the two will continue to play a major role in treatment for the foreseeable future.

An ounce of prevention being worth much more than a pound of cure, it behooves all of us to encourage our patients to stop smoking and stop ourselves before it is too late.

William L. MacVane, Jr., M.D.
211 State Street
Portland, Maine 04101

Anemia, Epigastric Pain and Anorexia in an 18-Year-Old Male

ALFRED E. SWETT, M.D.* and EUGENE P. MCMANAMY, M.D.**

The following case illustrates an unusual etiology of epigastric pain, anemia and vomiting in a young adult.

CASE REPORT

An 18-year-old white male was admitted to the Mercy Hospital with a complaint of epigastric pain. He states that he felt well until one week prior to admission. During this time, he has noticed intermittent epigastric pain which is non-radiating and daily vomiting of small amounts of whitish liquid. On further questioning, he admits to a gradual weight loss of 15 pounds during the past year.

Past history is unremarkable and there is no history of familial disease.

Physical examination shows a well-developed, emaciated, pale, white male with shoulder length hair. Vital signs are within normal limits. The pupils are round and equal and react to light and accommodation. The conjunctiva are pale and the eye grounds within normal limits. The nose and throat show no abnormality. There are small palpable nodes in the cervical region as well as within the axillae and inguinal region.

The examination of the chest was unremarkable. The abdomen is soft. There is a non-tender, movable, grapefruit size mass in the epigastrium somewhat to the left of the midline. Rectal examination is unremarkable.

Laboratory studies on admission were as follows: Hemoglobin, 6.2 gms.; Hematocrit, 23%; WBC, 11,000; 77 segs; 22 lymphs; 1 mono; platelets, 368,000. Glucose, 114 mg.; Uric Acid, 3.2 mg.; Cholesterol, 111 mg.; SGOT, 4 units; Total Protein, 6.7 gm.; Albumin, 4.0 gm.; LDH, 45 units; Alkaline Phosphatase, 37 units; Amylase, 49 units.

Urinalysis: 1.016 Specific Gravity. Clear, yellow. Negative sugar, albumin and occult blood.

Stool: Brown, formed with no evidence of occult blood or parasites.

The admission chest roentgenogram showed no abnormality. The scout radiograph of the abdomen shows a mass in the epigastrium and a normal spleen. The excretory urogram shows no abnormality of the urinary tract. The barium study of the upper gastrointestinal tract shows a very distended stomach filled with a large mass of reticulated material around which small amounts of barium slowly flow (Fig. 1). In addition, there is a large ulcer crater on the lesser curvature of the body of the stomach, (Fig. 2). A minimal quantity of barium also appears within the duodenum where there is further evidence of radiolucent filling defects.

A liver scan shows a large defect on the inferior hepatic margin corresponding to outline of the distended stomach.

On the following day, a surgical procedure was performed. At laparotomy, the stomach is large and firm. The gastric lumen is filled with firm, hard material which is found to be ingested hair in the form of a cast of the stomach (Fig. 3). This bolus which was found to extend into the proximal jejunum was removed intact. On the lesser curvature near the incisura is a large thick-walled, benign ulcer measuring one inch in diameter.

Fig. 1. Barium surrounds the large bolus of hair within the distended stomach.

The pathology report describes a trichobezoar which is a complete cast of the stomach and measures 30 x 5 x 15 cm. Attached to this is a cylindrical tail measuring 30 x 2 cm. In addition, a second specimen consists of a portion of stomach wall containing a deep and benign ulcer crater. Microscopic examination confirms this to be an active, benign, chronic peptic ulcer associated with a trichobezoar.

DISCUSSION

Bezoars are concretions of ingested material which are found in the gastrointestinal tract. The common variety of bezoar is formed from ingested hair or vegetable fibers. The first description of bezoar in the

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Fig. 2. In the frontal projection, the large ulcer crater on the lesser curvature is readily seen.

medical literature was made by Buaudamant in 1779. A survey of 172 cases of trichobezoars by Debakey and Ochsner reported 80% occurred in patients under 30 years, 90% were female and 40% in mentally disturbed patients.

The clinical manifestations include epigastric pain, occasional nausea and vomiting, diarrhea and constipation. The presence of an epigastric mass is commonplace. This patient exhibits a fairly typical clinical picture and has undoubtedly ingested his hair over a long period of time.

Complications of bezoar include obstruction (10.8%), ulceration (9.6%), hemorrhage (10%) and fistulas rarely.

Phytobezoar has been recently described as a complication of gastric surgery for ulcer. Treatment may be medical or surgical. Several methods of dissolving the hair and fiber concretions utilizing papain or multi-enzyme preparations have been described in the literature.

Fig. 3. The excised bezoar showing a cast of the stomach and the extension into the small intestine.

Summary

An unusual complication of the contemporary young male hair style is presented. The size of this bezoar caused nearly complete obstruction. In addition, a large gastric ulcer was also present.

References

1. Abourjaily, George S., et al: Multifocal trichobezoars of the gastrointestinal tract. Am. J. of Gastroenterology v. 47, 1967, p. 287.
2. DeBakey, M. and Ochsner, A.: Bezoars and Concretions: 303 cases. Surgery 4: 934, 1938.
3. Gellis, Sydney S. et al: Trichobezoars. Am. J. Dis. of Children: v. 123, #2, 1972.
4. Gwinn, J. L. and Barnes, G. R. Jr.: Trichobezoar. Am. J. Dis. of Children: 114: 311-312, 1967.
5. Margulis, A. R. and Burhenne, H. J.: Alimentary Tract Roentgenology. St. Louis, C. V. Mosby Co., p. 450.
6. Rogers, B. H. G. and Freimark, L. G.: Unusual Complications of Trichobezoar. Am. J. Dis. of Children: 110: 215, 1965.
7. Sanderson, Ian et al: Gastric phytobezoar following gastrectomy: Canadian Medical Journal v. 104, 1971, p. 1115.

Maine Heart Association Notes
20 Winter Street, Augusta, Maine

Systolic Time Intervals: A Non-Invasive Method to Determine Left Ventricular Function

Current Status, Advantages and Limitations

Ralph Shabetai, M.D.*

Measurement of systolic time intervals was developed as a technique to allow the clinician to assess the function of the left ventricle in a non-invasive manner. These intervals are obtained with the following simple instruments:

(1) A microphone placed on the precordium to register the first and second heart sounds distinctly.

(2) A transducer placed over the carotid artery to record its pulse contour with sharp onset and precise dicrotic notch.

(3) Conventional ECG electrodes to define the exact time of the onset of the QRS complex.

The three signals obtained with these devices are recorded on a three-channel strip chart recorder at a fast paper speed, preferably 100 mm/sec.

The left ventricle does not squeeze blood into the aorta slowly, as one squeezes juice from a lemon, but abruptly forces blood through its orifices. During isovolumic systole, a 50 millisecond period when the aortic and mitral valves are both closed, the ventricle develops tension. Near the time of peak tension, the aortic valve opens and the left ventricle rapidly ejects two-thirds of its content into the arterial system before the aortic valve again closes. Hemodynamic studies show that a normal ventricle, or a ventricle being stimulated pharmacologically, develops isovolumic tension more rapidly than a diseased ventricle, or one exposed to agents with negative inotropic effects. In addition, it has been demonstrated that left ventricular ejection time (LVET) varies directly with stroke volume. Thus, short isovolumic systole, large stroke volume, and long ejection time characterize normal left ventricular function.

LVET can be approximated easily by recording the time between the onset of ejection and dicrotic notch of the externally recorded carotid pulse. Weissler and associates calculated pre-ejection period by subtraction. They measured "total electromechanical systole" as the interval between the onset of the QRS complex and the first major component of the aortic valve closure sound. From total electromechanical systole, they subtracted the left ventricular ejection time determined from the external carotid pulse tracing. The difference yielded the pre-ejection period of systole (PEP). PEP includes the electromechanical coupling interval as well as isovolumic systole. However, hemodynamic studies have shown that PEP varies proportionately with isovolumic systole. Thus, Weissler's work divided systole into externally measured pre-ejection and ejection periods, and showed that normal ventricular function is characterized by a short pre-ejection period and a long ejection period. Not surprisingly, the ratio PEP/LVET is a more sensitive index of ventricular function than the numerator alone and, certainly, than the denominator alone.

Clearly, mechanical events in the heart and arteries are time dependent. Thus, the PEP associated with a fast heart rate is less than that seen with a slow rate, and LVET with a fast rate is less than with a slower rate, because tachycardia occurs largely, although not exclusively, at the expense of diastole. Weissler and his associates, using data from thousands of patients have developed regression equations from which normal PEP and LVET can be predicted as a function of heart rate. Other regression equations obtained show that PEP/LVET can be used to predict the ejection fraction, a sensitive ventriculographic index of left ventricular function.

Valvular disease alters the conditions affecting filling and emptying of the ventricles. Thus, systolic time intervals from patients with valvular disease are difficult to interpret. The technique has thus found its greatest application in detection and assessment of ischemic heart disease and myocardial disease. Systolic time intervals have also been employed to assess the effects of interventions, such as exercise, pacing, and drugs.

Measurement of systolic time intervals offered promise of becoming useful for serial study of left ventricular function, in patients in the coronary care unit after myocardial infarction, and in post-operative patients after coronary artery surgery. However, in both these areas, results have been conflicting.

How readily can these data be obtained? Reliable systolic time intervals can be recorded by any physi-

Continued on Page 142

*Department of Medicine, University of Texas Medical School, Houston, Texas 77025.
Prepared by the Maine Heart Association for this Journal.

From the Secretary's Notebook

Summary of Proceedings, Interim Meeting, M.M.A. House of Delegates, April 8, 1973 at Waterville, Maine

The Interim Meeting of the M.M.A. House of Delegates was held at Thayer Hospital in Waterville, Maine on Sunday, April 8, 1973 with an attendance of 48 delegates and alternates and three guests. George W. Wood, III, M.D., President of the M.M.A., called the meeting to order, and George W. Bostwick, M.D., Speaker of the House, presided.

1. **Financial Statement of Income and Expenditures for 1972 and Budget Proposed for 1974** was presented by Dr. Richard C. Leck, Chairman of the Budget Committee. Final action on the proposed budget will take place at the annual meeting of the House of Delegates on Saturday, June 16, 1973 at the Shawmut Inn, Kennebunkport, Maine.

2. The preliminary written **report of the Committee on Nominations** was presented. The report consisted of nominees for vacancies on the standing committees, and 2 nominees for the office of President-elect and for each position to be filled on the Executive Committee (this year, Districts 1, 5 and 6). **A brief biographical sketch of each nominee** was sent to members of the House of Delegates.

Final vote on the report of the Committee on Nominations will take place at the second session of the House of Delegates in June.

3. **Committee Reports:**

a) *Allied Health Professions* — William L. MacVane, Jr., M.D., Chairman, reported that this committee also serves as the physician-nurse liaison committee. They recently met with the nurses and discussed bills in the current legislature concerning allied health, physician assistants, etc. These bills were reviewed by the committee and some have apparently come up for hearing although the committee is not aware of what has happened to them and they have not heard from the Executive Committee about them.

Dr. Wood replied that the criticisms are valid and are being investigated by the Executive Committee. The Pediatric Nurse Associate Statement of Policy was reviewed by the Executive Committee and a motion is being proposed for its approval by the House of Delegates, Dr. Wood added. It was agreed that since this has just been received from the Committee and the delegates have not had a chance to review it, a decision on this will be postponed until June to give each county society an opportunity to review the statement more fully.

We have been in communication with our lobbyist re our direction on the P.A. bills, Dr. Wood added, and said that there are varied feelings about physician assistants and that the delegates should be thinking and arrive at a joint decision regarding them.

b) *Continuing Education* — A resolution was read by the Chairman, Dr. Richard T. Chamberlin, and it will be presented in June for action. Dr. Chamberlin added that he hoped to use the same form as the AMA uses for their Physician Recognition Award for collecting information. A discussion centered around whether a set number of hours should be required. It is Dr. Chamberlin's opinion that making it a requirement to report is as effective as actually requiring a certain number of hours.

c) *Diabetes* — Melvin Bacon, M.D., Chairman (copy given to each delegate).

d) *Peer Review* — Richard T. Chamberlin, M.D., Chairman (copy given to each delegate). Dr. Chamberlin added that the Peer Review Committee is still operating under the organizational EMCRO and that the request for an operational EMCRO was turned down. The Committee is now becoming involved in PSRO planning. In order to analyze State-wide aggregate data, the committee needs close to 100% data, and Dr. Chamberlin urged PAS hospitals to release data on PAS to their committee so it may join together with BCDS. The committee hopes to have a report in June on how this data can be used in peer review.

e) *Recruitment, Aid & Placement* — Dr. Robert E. McAfee, a member of this committee, reported for the chairman, Dr. Ferris S. Ray, that this committee has historically met once a year for the purpose of awarding loans under the Maine Medical Education Foundation. The committee recently discussed the distribution and balance of physicians in the State. They think a study should be done to ascertain the number of specialists in and required by each community, what the referral system is, what the ideal health care system is to attract and hold physicians in rural Maine, etc. The committee suggests this might be done via a questionnaire to each physician to see what they feel is needed. This idea was approved by the Executive Committee this morning, Dr. McAfee added. No action was taken by the House.

4. Several **resolutions** were presented and each

will be referred to the June meeting of the House of Delegates for action:

Committee on Continuing Education

It recommends that each member of the Maine Medical Association be required to report his activities in continuing medical education every year starting with 1 January 1974, according to a format provided by the Committee on Continuing Medical Education.

Penobscot County

Re Chiropractic in State Services — It recommends 1) That the State of Maine Department of Health and Welfare authorize treatment by chiropractors for any person for whom the Department provides services only when chiropractic treatment is ordered by a physician with the ordering physician to be responsible for the physical care of the patient in each case; so that he (the patient's **physician)** determine the effect of the chiropractic treatment for ordering its continuance under his (the physician's) management or for ordering its discontinuance; 2) That payment by the State of Maine Department of Health and Welfare or by its Division of Accounts and Audits for chiropractic services (for persons receiving services from the Department) be made only when the chiropractic services have been ordered by a physician — the Department to determine whether the chiropractic services have been ordered by a physician and that the patient is under the care of the ordering physician in each case.

Dr. Benoit Ouellette, Aroostook County

It recommends that the Association vote for acceptance and participation of the PSRO regimentation with complete submission, or vote a non compliance to the unworkable concept of PSRO and force the government to recourse some acceptable bargain with the Association.

5. Other business —

a) **Chiropractic** — Legislative bills on chiropractic were discussed, and Dr. Wood brought the delegates up to date on the Association's activities in this area and urged vigorous action from all the physicians. Dr. Hanley further urged that every possible physician, and their friends, attend a hearing on L.D. 1258, a bill to eliminate chiropractic in Maine, at the Augusta Civic Center on April 25th.

b) Dr. Earl Wedrow of Boston reported that the Massachusetts Medical Society has organized a group of 20 speakers to give **continuing education** courses and they would be willing to come into Maine.

c) Dr. Wood reported that a meeting was held to discuss hiring **additional personnel** at the M.M.A. headquarters. The plan is to seek out a sharp young individual, non-MD, to assist Dr. Hanley, and a job description will be brought to the June meeting.

PATRICIA A. BERGERON
Secretary-Treasurer, M.M.A.

Meetings of Medical Interest —

Annual Meeting of the Maine Chapter, American Academy of Family Physicians, Howard Johnson Motor Inn, Exit 8, South Portland — December 1

AMA Clinical Convention, Anaheim, California — December 1-5

ANNUAL MEETING HELD

At the Maine Blue Cross and Blue Shield Annual Meeting held Wednesday, April 25, 1973, Owens S. Estey and Lloyd H. Holmes were elected to the Board of Directors.

Mr. Estey, of Mapleton, replaces Oscar Benn as a director representing subscribing members. He is also a member of Maine Blue Cross and Blue Shield Consumer Advisory Council and was appointed by the Governor as a member of the "Mark Maine" Committee.

Mr. Holmes will be the new director representing participating hospitals, replacing Mr. Theodore Fobes. A trustee of the Osteopathic Hospital of Maine, Holmes is also Vice-President of the Board of Directors of Junior Achievement. He lives in Portland.

Both Benn and Fobes are retiring from their directorships. Six directors were reelected to serve another 3-year term. They are Robert W. Hudson, Adelbert M. Jakeman, Louis A. Asali, M.D., Daniel F. Hanley, M.D., Woodrow Page, and Joseph S. Jones.

The 1972 Maine Blue Cross and Blue Shield Annual Report was made public at the Annual Meeting. It shows impressive gains in membership, which increased to over 444,000 Maine residents, and an operational expense of only 7.6% of the total income. The underlying reasons for the obvious success of the organization are at the crux of the report.

The report shows that Maine Blue Cross and Blue Shield is unique in a number of ways, including its contracts with hospitals and physicians, the company's involvement in health planning and other professional organizations, its commitment to the consumer through consumer councils, health care education, and development of new benefit programs to better utilize existing health care facilities.

One of the new benefit programs made available during 1972 is Coordinated Home Healthcare. This program lets patients in a participating Blue Cross hospital go home with his physician's permission to recuperate under a regular plan of treatment designed specifically to provide the medical and nursing care he needs to recover. It serves a two-fold purpose by aiding the patient in his home and helping to control the cost of care. It produces savings without any sacrifice in the quality of treatment.

Maine Blue Cross and Blue Shield also established a new department in 1972, Healthcare Planning and Research, to meet the expanding needs in the complex process surrounding the study and development of new programs. This new department examines benefit programs to assure their effectiveness and, in cooperation with providers and Health Planning Agencies, will research and develop new ways to provide benefits.

Maine Blue Cross and Blue Shield has taken the initiative in soliciting consumer advice. In 1972, representatives of the four Regional Consumer Advisory Councils were invited to visit the Portland home office to study and comment on the entire operation. Also, Blue Cross and Blue Shield Representatives have been in attendance at the Consumer meetings to provide information when needed and to benefit from the consumer point of view.

At the Annual Meeting, the Maine Blue Cross and Blue Shield Board of Directors also elected John Mitchell as clerk and appointed him General Counsel. An amendment to the bylaws to eliminate the now vacant position of Executive Vice-President was passed since recent administrative changes have removed the need for this position.

News, Notes and Announcements

American Board of Family Practice Announces Examination Dates

The American Board of Family Practice announces that it will give its next two-day written certification examination on October 20-21, 1973 in various centers throughout the United States. Information regarding the examination can be obtained by writing:

Nicholas J. Pisacano, M.D., Secretary
American Board of Family Practice, Inc.
University of Kentucky Medical Center
Annex No. 2, Room 229
Lexington, Kentucky 40506

PLEASE NOTE: It is necessary for each physician desiring to take the examination to file a completed application with the Board office. *Deadline for receipt of applications in the Board office is August 1, 1973.*

Annual Otolaryngologic Assembly

October 20 through 26, 1973

The Annual Otolaryngologic Assembly of 1973 will be held October 20 through 26, 1973, in the Eye and Ear Infirmary of the University of Illinois Hospital. The Department of Otolaryngology of the Abraham Lincoln School of Medicine, University of Illinois at the Medical Center, offers a condensed basic and clinical program for practicing otolaryngologists under the direction of Emanuel M. Skolnik, M.D., with Burton J. Soboroff, M.D., as co-chairman. This program is designed to bring to specialists current information in medical and surgical otorhinolaryngology.

Interested otolaryngologists should direct their inquiries to the mailing address: Otolaryngology, P.O. Box 6998, Chicago, Ill. 60680.

A separate, but correlated course, "Conference on Radiology in Otolaryngology and Ophthalmology" will be held this year on Friday and Saturday, November 23 and 24, under the guidance of Galdino E. Valvassori, M D. For further information about the radiology conference, write to Professor Valvassori, Radiology Department, Abraham Lincoln School of Medicine, P.O. Box 6998, Chicago, Ill. 60680.

National Conference on Virology and Immunology in Human Cancer

November 29, 1973 — December 1, 1973

Waldorf-Astoria Hotel
New York, New York

Sponsored by: American Cancer Society
National Cancer Institute

The purpose of this Conference is to present to the medical and related professions the current developments in research and clinical investigation in virology and immunology and the assessment and implications of this work in the prevention and treatment of human cancer.

Sessions are open to all members of the medical and related health professions. Pre-registration is requested. There is no registration fee.

For information write: Sidney L. Arje, M.D., National Conference on Virology and Immunology in Human Cancer, American Cancer Society, Inc., 219 East 42nd Street, New York, New York 10017.

University of California School of Public Health, Berkeley Announces Postgraduate Programs for Physicians

The Maternal and Child Health Program of the University of California School of Public Health at Berkeley announces its postgraduate programs for physicians. These programs all lead to the degree of Master of Public Health. *Tax-exempt* Fellowship support is available. Applications are now being accepted for September *1974*.

Maternal and Child Health. A nine-month academic program in planning, organizing and operating comprehensive health services for mothers and children.

Family Planning. A nine-month academic program providing intensive work in family planning as part of the general graduate preparation in maternal health, and maternal and child health.

School Health and Adolescence. A nine-month academic program providing intensive work in school health and adolescent medicine as part of the general graduate preparation in maternal and child health.

The Multiple Handicapped and Mentally Retarded Child. A twenty-one month academic and clinical program in planning, organizing and operating community services for children with multiple handicaps, including mental retardation. This program is primarily for Board-Certified or Board-Eligible pediatricians.

Comprehensive Health Care. A twenty-one month academic and clinical program in planning, organizing and operating programs for the delivery of comprehensive health and related care to mothers, children and families. This program is primarily for Board-Certified or Board-Eligible pediatricians and obstetricians.

Perinatology. A twenty-one month academic and clinical program combining nine months in the School of Public Health with 12 months of full-time fellowship in Perinatology, including the prenatal, natal and postnatal phases. This program is primarily for Board-Certified or Board-Eligible pediatricians.

For information, write to Helen M. Wallace, M.D., Professor and Chairman, Maternal and Child Health Program, School of Public Health, University of California, Berkeley, California 94720.

MediPhone 24 Hour-a-day Medical Telephone Consultation

MediPhone is a direct *nationwide medical* telephone consultation service available to all physicians — *24 hours a day, everyday.*

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World Health Organization Fellowships Available To United States Health Workers

The World Health Organization will make available in 1974 a limited number of short-term fellowships for travel abroad related to the "improvement and expansion of health services" in the United States. This support is to United States citizens engaged in operational or educational aspects of public health employed by governmental (non-Federal) agencies or educational institutions. In selecting applications, a special committee will consider the professional background of the individual, the field and locale of the study proposed, and the utilization of the experience by the applicant on his return. Employees of the Federal Government are not eligible. Applications will not be considered for the pursuit of pure research projects, for attendance at international meetings, nor from students in the midst of training at either the undergraduate or graduate level. Applicants may not be more than 55 years of age.

A fellowship award will cover per diem and transportation. Except in very unusual circumstances, it will be limited to short-term travel programs averaging about two months. Employers of successful applicants will be expected to endorse applications and to continue salary during the fellowships.

Priorities of award will be established up to the total of the funds available. The deadline for the receipt of completed applications is September 30, 1973.

Further information may be obtained from Dr. Robert W. Jones, III, Chief, International Education Branch, Fogarty International Center, National Institutes of Health, Room 2B-55, Building 31, Bethesda, Maryland 20014.

State of Maine Board of Registration of Medicine Secretary — George E. Sullivan, M.D. Waterville, Maine

Physicians Licensed to Practice Medicine and Surgery in the State of Maine

Through Reciprocity

Bach, Robert D.; Collins, Thomas, D.; Hoehn, James L.; Jollie, Peter M. O.; Kurtz, Kenneth J.; Langley, James W.; Nevius, John K., Jr.; Seelig, Michael J.; Swan, Davis M.; Cote, Robert P.; Roberts, Lloyd; Smith, Alan F.; Record, Nelson B., Jr.; McCabe, George E.; and Leong, Keng-Cheong.

Fifty Training Programs in Clinical Pharmacology Described in New Publication

The first detailed guide to training programs in clinical pharmacology — the study of drugs in man — has just been published by Pfizer Inc.

The 105-page booklet, entitled "Clinical Pharmacology — A Guide to Training Programs," describes the 50 programs offered throughout the United States and Canada. Included are the clinical focus of each program, the facilities and training support available to the student, a listing of the faculty, a description of the school and application procedures.

Noting in the preface that the demand for specialists in clinical pharmacology exceeds the supply all over the world, Dr. Sheldon Gilgore, President of Pfizer Pharmaceuticals, states that the booklet is a guide for "young physicians who are looking for the challenge that clinical pharmacology will increasingly offer, both as a scientific discipline and as a very direct means of service to society."

Dr. Louis Lasagna, Director of the Department of Pharmacology and Toxicology, University of Rochester School of Medicine, writes in the introduction that well-trained clinical pharmacologists are "desperately needed" to help discover and evaluate new drugs, and that this booklet will provide "invaluable help for those who contemplate careers in this exciting discipline."

The American Society for Pharmacology and Experimental Therapeutics points out in introductory remarks that it "is pleased to see a new compendium of active training programs in clinical pharmacology" and that "the Society stands ready to encourage and help prospective clinical pharmacologists as they explore what is perhaps the most exciting area of basic science that directly relates and applies to man."

Descriptions of the various programs are written by the directors. Items covered in the booklet include names of recent graduates and their career choices as well as a selection of staff publications. It also contains a separate section on sources of support for trainees in clinical pharmacology.

Copies of the booklet will be made available to medical schools for distribution to physicians interested in pursuing a career in clinical pharmacology. A limited number of copies also are available to physicians from the Pfizer Public Affairs Division, 235 East 42nd St., New York, N.Y. 10017.

American Cancer Society's Second National Conference on Cancer of the Colon and Rectum

September 27-29
Americana Hotel
Bal Harbour, Florida

This conference will present *up-dated* information by leading authorities in epidemiology, pathogenesis, etiology, host factors, detection, diagnosis, treatment and rehabilitation in cancer of the colon and rectum.

Sessions are open to all members of the medical and related health professions. Pre-registration is requested. There is no registration fee.

Accredited by the American Medical Association Council on Medical Education.

For information write: Sidney L. Arje, M.D., Second National Conference on Cancer of the Colon and Rectum, c/o American Cancer Society, 219 East 42nd Street, New York, New York 10017.

WANTED

Issues of the Maine Pharmaceutical Association *Proceedings*, 1867-1905. Desperately needed by the Portland Public Library, 619 Congress Street, Portland, Maine 04101. Original copies were unfortunately lost by the bindery. Have historical value tracing development of pharmacy in Maine.

Contact: Mrs. Barbara Smith, Assistant Director, Portland Public Library. 207-773-4761.

County Society Notes

KENNEBEC

The Kennebec County Medical Association met at the Holiday Inn in Augusta, Maine on February 15, 1973. Following a satisfactory roast beef dinner, the President, Dr. Richard R. Dole, presided over a brief business meeting.

The minutes of the last meeting were accepted as read by the secretary.

A letter from Mr. John E. Fickett, Director, Division of Medical Assistance, Department of Health and Welfare, was read. This letter called attention to the "Early and Periodic Screening, Diagnosis and Treatment" program which has been instituted as part of Title XIX services in Maine. A fact sheet on the program was distributed to the members present.

Dr. Richard Giuffre of Winthrop was elected to membership and was welcomed by the President and members present.

The application for membership of Dr. Kenneth W. Green of Waterville was announced. Dr. Green is presently in the Department of Anesthesia at Seton Hospital, Waterville.

The speaker of the evening was Dr. Loring W. Pratt, who spoke on the "Rationale for Surgery in Head and Neck Malignancy." In his informative and well-illustrated lecture, Dr. Pratt discussed the indications for surgery in terms of 1) Biopsy; 2) Curative; 3) Palliative; 4) Neurologic and 5) Reconstructive Procedures.

The meeting was adjourned at 9:45 p.m.

The Kennebec County Medical Association met at the Silent Woman Restaurant in Waterville, Maine on March 15, 1973. Following the social hour and dinner, the President, Dr. Richard Dole called the meeting to order.

Minutes of the previous meeting were accepted as read by the Secretary. Several communications were read. First, a letter from the Maine Medical Association notifying the county association of two Association members to be honored at the June meeting of the Maine Medical Association. Dr. Ralph L. Reynolds will receive his 60-year pin and Dr. Howard F. Hill will receive his 50-year pin. A letter from Mr. Joseph Hasenfus regarding the SAMA-MECO program for summer medical students was read. The request for financial support by the Association for this program would be considered a decision rendered at the next meeting. An announcement of the Woman's Auxiliary to the Kennebec County Medical Association program to benefit the medical careers scholarship fund was made. This event will be held on Saturday, April 28 at the Gardiner Lions Club on Brunswick Road. The occasion is a dinner-dance and the members were urged to attend this occasion.

The application for membership of Dr. Murrill Szucs, Jr. of Waterville was announced. Dr. Szucs is presently practicing internal medicine in Waterville.

Dr. Kenneth Green of Waterville was elected to membership and welcomed by the President and by the members present.

Under old business, a report of the Ad Hoc Medical-Legal Committee meeting of February 26 was read. Dr. William E. Schumacher, chairman of the Bylaws committee asked for an expression of the membership regarding the revision of the Bylaws, especially with regard to peer review. Some informal discussion followed and Dr. Dole announced that Dr. Richard T. Chamberlin will be asked to speak on the subject of peer review at the next meeting. Dr. Schumacher will, in the meantime, send copies of the present Bylaws to all members of the Association to serve as a base line for discussion. Dr. Loring W. Pratt announced the availability of a new treatment for cleft palate diagnosed at birth. This new method involves the fitting of an artificial palate in the first 12 hours of the infant's life. He stressed the importance of the early recognition and treatment of this condition and announced that this would be more fully described in *The Journal of the Maine Medical Association.*

The speaker of the evening was Dr. Peter Yurchak, Assistant Professor in Medicine of Harvard Medical School and member of the Cardiology Unit of the Massachusetts General Hospital. Dr. Yurchak gave a most lucid and interesting presentation of selected problems of congenital heart disease in the adult.

The meeting was adjourned at 9:50 p.m.

The Kennebec County Medical Association met at the Jefferson Restaurant in Waterville, Maine on April 19, 1973. Following a social hour and dinner, the President, Dr. Richard R. Dole called the meeting to order. The minutes of the previous meeting were accepted as read by the Secretary.

Communications: A letter from the A.M.A. concerning the International Conference on Environmental Health to be held in Yugoslavia in October was read. A letter from Mrs. Twadelle requesting a contribution to the Woman's Auxiliary Health Careers Scholarship Fund was read. The Secretary was directed to check the records to see what our last contribution was and to make the same contribution for this year.

Murrill Szucs, M.D. was elected to membership in the Association.

Old business: The request of Mr. Hasenfus for the Association to contribute funds for the S.A.M.A. summer student program was discussed. Dr. John T. Chen moved that the Association not contribute funds for this purpose and the motion was carried. It was suggested that the Secretary recommend to Mr. Hasenfus that the Woman's Auxiliary might be approached for support. Dr. William E. Schumacher reported on the activity of the Bylaws Committee and Dr. Dole directed the committee to proceed with the formulation of a new set of bylaws for presentation to the Association. Dr. Richard T. Chamberlin reported on the legislation proposed by Senator McLeod to repeal the chiropractic practice act and urged widespread support at the hearing to be held on April 25 in Augusta.

New business: The resolution to establish an affiliate membership for hardship cases which would exempt the member from paying dues was presented and discussed. Dr. Schumacher moved that the resolution be revised to add the phrase "For other compelling reasons" to the wording of the section in the bylaws of the Maine Medical Association concerning affiliate membership. This motion was carried.

Dr. Dole then introduced the speaker of the evening, Richard A. R. Fraser, M.D., who gave a lucid presentation of the Surgical Management of Intracranial Aneurysms. The high possibility or rebleeding from an intracranial aneurysm over the succeeding three to five years, the technical advances in the surgical approach to these lesions utilizing microscopy, hypotension and postoperative angiography renders the definitive management of intracranial aneurysms preferable, in most cases, to more conservative medical therapy. Dr. Fraser's talk was well received by the approximately 45 members present. The meeting was adjourned at 9:50 p.m.

KEVIN HILL, M.D., *Secretary*

LINCOLN-SAGADAHOC

A regular monthly meeting of the Lincoln-Sagadahoc County Medical Society was held at The Ledges in Wiscasset, Maine on March 20, 1973

The meeting was called to order at 8:56 p.m. by the President, Dr. Peter A. Evans. The minutes of the February meeting were read and accepted as read.

There being no committee reports, old business, or new business, Dr. Evans turned the meeting over to Dr. Louis Bachrach, Chairman of the Program Committee, who introduced Dr. Gerold K. V. Klein of Brunswick. Dr. Klein spoke on "Bromelain in the Treatment of Burns."

The meeting was adjourned at 10:30 p.m.

A regular monthly meeting of the Lincoln-Sagadahoc County Medical Society was held at The Ledges in Wiscasset, Maine on April 17, 1973.

The meeting was called to order at 8:40 p.m. by the President, Dr. Peter A. Evans. The minutes of the last meeting were read and accepted as read.

There was no old business.

New business: Dr. Richard C. Leck discussed the major items of discussion at last week's meetings of the M.M.A. Executive Committee and House of Delegates: The proposed 1974 budget, the question of an Assistant Executive Director, PSRO for the State and the Regional Blood Bank. Dr. Paul A. Fichtner spoke of Legislative Document No. 1258, a proposal to limit health services to scientific practice; a hearing will be held at the Augusta Civic Center, and all physicians are urged to attend in force to second passage of this proposed law, on 25 April. Dr Bostwick mentioned also, a recommendation of the Committee on Continuing Education that members be required to report hours spent in acquiring postgraduate education. Heated discussion of PSRO followed. Dr. Fichtner announced that rates for the M.M.A. group Blue Cross-Blue Shield contract are to rise.

Dr. Louis Bachrach introduced Dr. Leith Hartmann, who spoke on military medicine in Viet Nam. Many interesting slides were shown.

The meeting was adjourned at 10:25 p.m.

GEORGE W. BOSTWICK, M.D., *Secretary*

CUMBERLAND

The 376th meeting of the Cumberland County Medical Society was held on March 15, 1973 at the Holiday Inn on Riverside Street in Portland, Maine. Sixty members were in attendance. Following the social hour, dinner was served. Both the dinner and the service were outstanding for their lack of quality.

The business meeting was called to order by the President, Dr. Douglas R. Hill at 8:30 p.m. Reading of the minutes of the last meeting was omitted. Following a second reading of applications, Drs. Thomas A. Martin, Jr. and Cathel McLeod were voted into active membership. The first reading of Dr. Fred Ginn's application was made.

The following members will receive honorary pins at the annual meeting in June: Fifty-year pins for Drs. Abram I. Cohen, Eugene E. O'Donnell and Theodore M. Stevens; Fifty-five-year pin for Dr. William E. Freeman; and Sixty-year pins for Drs. Jacob Melnick and Albert W. Moulton, Sr.

Dr. Hill named the following members of the Nominating Committee: Drs. Lawrence Crane, Chairman; Philip G. Whitney, Ronald J. Carroll, Kirk K. Barnes and Morrill Shapiro.

There will be a meeting of the Maine Medical Association House of Delegates on April 8th in Waterville. Delegates and alternates are urged to attend.

A resolution on the death of Dr. Harold E. Libby of Westbrook was presented by Dr. Sidney R. Branson. This will be entered into our records.

Many favorable comments concerning our television show have been received. Our second presentation will include Drs. Ronald Carroll, Clement A. Hiebert, William L. MacVane, Jr. and Darrell P. Thorpe.

An offer from CIBA Pharmaceutical to sponsor a free hypertensive clinic in the Portland area was accepted.

Following a very lengthy discussion and much controversy, a motion to provide an informational telephone service was seconded and approved. This will be done on a 9 to 5 basis and will be handled by an answering service on a trial basis for 6 months to one year.

Dr. Hill announced the April meeting at the new Holiday Inn in Portland on April 24, a Tuesday. At this meeting, Dr. Raymond Holden will be our guest speaker.

Financial support for a summer program for medical students sponsored by SAMA was requested by Dr. MacVane since Regional Medical Program funds have been cut. Following a lengthy discussion, $100 was approved as a contribution to this fund.

Dr. Hill further announced that Drs. George F. Sager and Ronald Carroll will represent Cumberland County on the State Peer Review Committee. Dr. Charles W. Capron reported for the Cumberland County Legislative Committee. The Maine Medical Association now has a full-time lobbyist, Mr. Charles Cragin, III from the law firm of Verrill, Dana and Philbrick, representing us in Augusta. Weekly reports are being received by all County Societies.

There being no further new business, the meeting was adjourned at 10:00 p.m.

ALFRED E. SWETT, M.D., *Secretary*

Letter to the Editor

To the Editor:

A Venture in Education
Workshop on Stroke

For many years, I have been interested in Professional and Non-Professional Education Programs in York County. These have been in the form of Institutes for Nurses in York County in a variety of fields. These have all been short programs. However, a day long program was developed on "Stroke" and was held at Nasson College, Springvale, Maine on April 3, 1973 from 8:30 a.m. to 4:00 p.m. It was a combined project of the Maine Heart Association and the Sanford-Springvale Community Health Association. It had the approval of the Maine Medical Association and the York County Medical Society. It was for R.N.s, L.P.N.s and nursing students. The results, as far as we were concerned, were superb. Almost two hundred from these three categories attended. A certificate of attendance will be sent to all the R.N.s, L.P.N.s and students who attended. Because of the success achieved, it was deemed of interest to list the speakers and their subjects: Charles F. Branch, M.D. on "Epidemiology, Etiology and Pathology of Stroke;" Jacob Dana, M.D. on "Diagnosis and Laboratory Procedures in Stroke;" Joseph H. Edgar, Jr., M.D. on "The Medical Management of the Stroke Patient;" Carl Scholl on "The Role of Physiotherapy in Stroke;" Sandy Rousseau on "Occupational Therapy in the Stroke Patient;" Elaine R. Stevens, M.A. on "Speech Therapy in the Stroke Patient;" Nicholas Fish, M.D. on "The Psychological Aspects of the Stroke Patient;" Carolyn Russell, R.D. on "The Dietary Program in the Stroke Patient;" Katherine Greeley on "The Role of Social Service in the Stroke Patient;" Kathy Kaulakis, R.N. on "The Nursing Care of the Hospitalized Stroke Patient" and Virginia Bundy, R.N. on "The Nursing Home Care of the Stroke Patient."

There was a five-minute question and answer period following each talk.

Participants come from all over the State. We believe there should be more of this type Educational Programs throughout the State, and we encourage other County Societies to sponsor them.

May I close with this dictum, "If you live a life of usefulness trying always to help others, you need not seek happiness . . . it will come with each rising sun."

N.B. I am dedicating this meeting to my mother who died of a stroke; two of her brothers and one sister also died of a stroke as well as her mother and father. This clearly indicates the familial tendency in this condition.

May I thank Mrs. Adelaide Koontz of the Maine Heart Association and my secretary for the help given me and also to Nasson College, Springvale, Maine for allowing us to use their facilities.

MELVIN BACON, M.D.
Member, Board of Directors, Maine Heart Association; Chairman, Medical Advisory Committee, Sanford-Springvale Community Health Association

SYSTOLIC TIME INTERVALS: A NON-INVASIVE METHOD TO DETERMINE LEFT VENTRICULAR FUNCTION — *Continued from Page* 134

cian who has ready access to a high-quality multichannel phonocardiograph, performs the technique frequently, is careful to assure high quality ECG, phonocardiogram and pulse tracings, has the time and patience to make accurate measurements, and understands the limitations of the method.

Can systolic time interval measurement ever substitute for cardiac catheterization to yield information that is not apparent from the history, physical examination, ECG and chest X-ray? For a decade or more, the systolic time interval technique gained little acceptance and was not utilized by most cardiologists. As the need to evaluate left ventricular function increased, systolic time intervals became more popular. Unfortunately, the technique was frequently employed in a manner which its originators would deplore.

Measurement of systolic time intervals seldom yields data that surprise the experienced cardiologist. When considered in context, such measurements may supply useful information. Moreover, they have two cardinal virtues:

(1) They can be performed repeatedly with no danger, little or no discomfort, and little expense to the patient; and

(2) The results are quantitative. Thus, serial changes can be detected readily. Accordingly, measurement of systolic time intervals is likely to play an increasing role in clinical cardiology and deserves continued energetic research effort.

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The Journal
of the
Maine Medical Association

Volume Sixty-four | Brunswick, Maine, July 1973 | Number 7

Anti-Sd[a]

A Problem in Cross-Match of Blood

A Case Report

ELIZABETH J. HAUSMAN, B.S., MT(ASCP)*

Difficulties in cross-matching can occur in any of the antigen-antibody systems, such as M, N, P, and Le. This report describes the difficulties encountered due to the Sd[a] antibody and reviews the literature.

CASE REPORT

A 46-year-old white male was admitted to the Veterans Administration Center, Togus, as a transfer from another VA hospital for further treatment of Laennec's cirrhosis. His laboratory tests revealed the following values: Initial hematocrit, 28%, subsequently dropping to 17%. Attempts to cross-match the patient proved futile because his blood was incompatible on both the major and minor sides with type-specific blood. The patient's chart indicated that he had received one pint of type-specific blood at the Fresno, California, VA Hospital, where the presence of a strong cold agglutinin was noted. The patient received the blood without incident. Other laboratory studies disclosed that the patient had a high bilirubin value, a reverse A/G ratio, and an abnormal bleeding profile.

Due to the fact that the patient could not be transfused at this Center, he was transferred to the VA Hospital, Boston, Mass., where they attempted to cross-match him with many units of blood all of which showed an incompatibility. A sample of his blood was sent to the National Red Cross Rare Antibody Laboratory in Washington, D. C., where it was reported that the patient had several antibodies: An anti-I, an anti-H (at 4° C. only), an anti-Sd[a], and there was a question as to whether the patient might have an anti-K (See Table 1). The patient's sister was found to be compatible and gave him one pint of blood. The patient was transferred back to this Center where he rapidly deteriorated and died June 12, 1972, from hepatorenal failure.

TABLE 1

PATIENT'S GENOTYPE

ANTI-A	∘	ANTI-C	+	
ANTI-B	+	ANTI-E	∘	
ANTI-D	+	ANTI-c(hr')	+	
A_1 CELLS	+	ANTI-e(hr'')	+	
B CELLS	∘	ANTI-K	∘	
ANTI-M	+	ANTI-k	+	
ANTI-N	+	ANTI-Fy[a] +	Fy[b]	∘
ANTI-S	∘	ANTI-Le[a] ∘	Le[b]	∘
ANTI-s	+	ANTI-Jk[a] +	Jk[b]	∘
ANTI-P_1	+	ANTI-Lu[a] ∘	Lu[b]	+
		ANTI-Sd[a]	∘	

DISCUSSION

The Sd[a] antigen-antibody system has been a problem since 1956 when it was described for the first time. It creates a problem on cross-matches because of the variation in strength of the reactions. Approximately 1% of the population are strong reactors and show massive agglutination. Another 80% of the population show moderate agglutination. The major problem occurs in the 10% of the population that produce only a weak agglutination and show only an occasional clump of cells among free-flowing cells. Many of these people will react with only the strongest antisera and even this test may need to be repeated before one can determine if they are positive or negative.

Most reliable results for a grouping technique are obtained using a 2% to 4% saline suspension

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of red cells with antisera in tubes and incubating for 1 to 1½ hours. The cells are put on a slide and allowed to stand for 30 to 60 minutes in a moist chamber with an occasional rocking. The strong reactors will become apparent immediately but the weaker ones will show the typical agglutination pattern only after incubation for one hour.[1]

The other problem with the Sd^a blood factor is the fact that the agglutination pattern is very similar to the Lu^a antibody.

This blood factor was named after the blood donor in whom it was first found and was called Sid. It is referred to also as the Hay-Holmes or Kirby antibody. Approximately 91% of Caucasian people have the Sd^a antigen. All cord bloods that have been tested are Sd^a negative.[2] The antigen does not manifest itself until about the age of seven months (one exception was a ten-week-old infant).[1]

The antibody is biochemically an IgM complement-fixing antibody and has been found to occur in the sera of donors who have not had any previous stimulation such as transfusions or pregnancy. It is picked up easily on Coombs cross-match because of its complement-fixing ability. Most antiglobulin sera used have a weak anti-gamma M component; However, if a high titer of anti-gamma M reagent is used, anti-Sd^a will be found in the absence of complement. If there is an absorption of complement to the antibody coated cells, there could be an interference with the ability of the anti-gamma M to agglutinate them. The antibody will agglutinate a saline suspension of red cells at 20° C. and usually will agglutinate the red cells at 37° C. Sometimes the reactions will be weaker at higher temperatures. Red cells that were treated with papain showed hemolysis in some of the serum samples.[3]

Adsorption studies showed that the strong reactors will absorb the antibody very easily whereas the weak reactors absorb only minute traces of antibody and are difficult to differentiate from the negative ones.[1]

With regard to the best means of detecting the antibody, use of a saline suspension of red cells at 16° and 37° C. has been effective but agglutination was difficult to observe because of the small proportion of cells agglutinated. The use of albumin at 37° C. did not enhance the agglutination.

Good results were obtained by enzyme treatment of cells with papain and ficin. Ficin, in addition, eliminates the false positive since non-specific complement fixation is overcome. The best results were obtained by using an anti-human globulin technique with anti-globulin reagents that contained either a potent anti-gamma M or an anti-complement antibody.

Although Sd^a has not been implicated directly in a transfusion reaction, it is of practical importance because occasionally it may be detected in the sera of individuals thus presenting a problem in cross-matching. The clinical importance of Sd^a is still uncertain. The fact that it is a complement-fixing antibody, in addition to being occasionally hemolytic in vitro, suggests that it could accelerate destruction of transfused red cells.

A case has been reported where an individual had a strong Sd^a antibody in his serum and showed no untoward reaction after receiving a large quantity of Sd positive blood.[3]

REFERENCES

1. Macvie, S. I., Morton, J. A., and Pickles, M. M.: The reactions and inheritance of a new blood group antigen, Sd^a. Vox Sang. 13: 485-492, Dec., 1967.
2. Race, R. R., and Sanger, Ruth: Blood Groups in Man, 5th ed. Philadelphia: F. A. Davis Co., 1968.
3. Renton, H., et al.: Anti-Sd^a, a new blood antibody. Vox Sang. 13: 493-501, Dec., 1967.

Meetings of Medical Interest –

Annual Meeting of the Maine Chapter, American Academy of Family Physicians, Howard Johnson Motor Inn, Exit 8, South Portland — December 1

AMA Clinical Convention, Anaheim, California — December 1-5

Dual Stressed Retention Sutures

DONALD B. HAWKINS, M.D.*

The usual retention sutures have several well known disadvantages. Among these are: Cutting of the skin at the points of entrance and exit of the suture, local infection at these sites, patient discomfort, and poor cosmetic result.

This description is of a method which obviates most of these disadvantages. Hopefully, the illustrations will for the most part be self-explanatory.

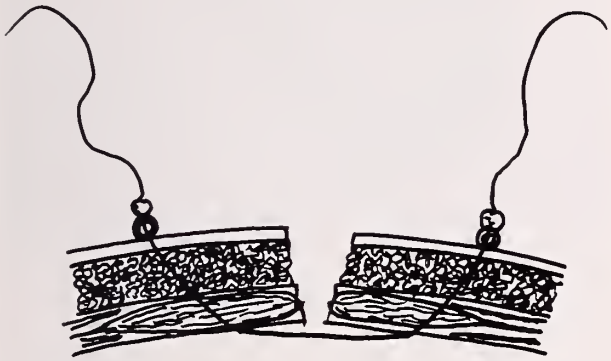

Fig. 1

The general principal of the method is illustrated in Fig. 1. The sutures are passed through pieces of soft rubber tubing, ⅜ to ½ inch in diameter, and roughly the length of the wound, and placed parallel to it on either side. They are then snugged up individually, and "locked" above the tubing by a compressed split lead shot each side. This allows for close approximation of the lower layers of the wound without compressing and strangulating the upper layers, and the strain is then distributed along the wound parallel to the incision rather than transversely across it. The tubing places this strain evenly along the sides of the wound avoiding points of excessive pressure. The sutures are then tied across the incision with only sufficient tension to approximate the upper layers. The "dual stressed" principal is illustrated by the heavy and light arrows in Fig. 2. That portion of the suture lying under the tubing and approximating the lowermost portions of the wound is under the greatest stress, and that above the skin is under the least stress.

It is of some importance that an effort be made to place the points of entrance and exit of the sutures at about the same distance from the edges of the incision.

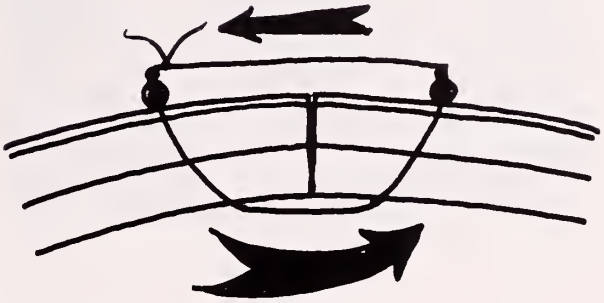

Fig. 2

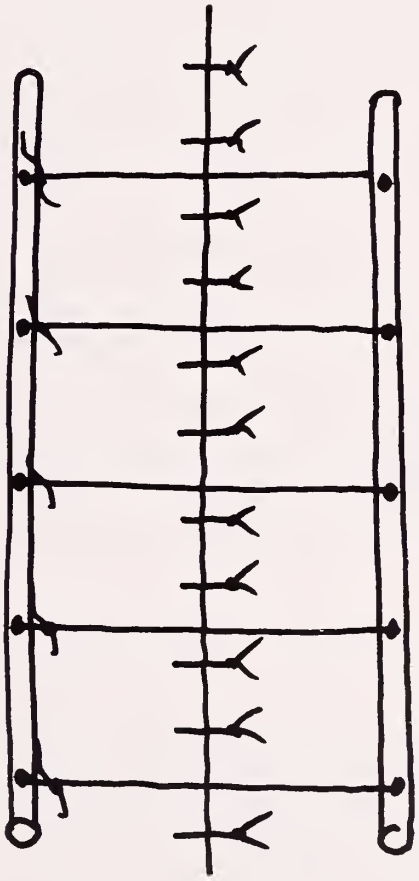

Fig. 3

Individual skin sutures may be placed, if desired, either before or after tieing the upper portion of the retention suture. Finally, a strip of sterile gauze may be slid under the tied sutures, over the wound. The finished closure appears as in Fig. 3.

*Staff Physician, Veterans Administration Center, Togus, Maine 04330.

Modification of Plastic Culture Dishes for Single Cell Observations

LUCIAN J. CUPRAK, D.M.D., PH.D.* and ROBERT L. STAHL**

INTRODUCTION

The determination of the essential nutritional requirements of cells in culture has been done with either the monolayer or suspension culture techniques.[1] These methods usually employ replicate cultures with a large initial cell inoculum (10^4-10^5 cells) and necessitate frequent estimations of the cell number or total cellular protein of at least duplicate cultures over a period of time sufficient to establish a population doubling time. For example, Eagle[2] had tested each nutrient of a basal medium by its single omission and judged essentiality by noting the effect of the single omission upon the growth of a clonal cell culture. The length of time required to demonstrate certain nutritional requirements often makes it necessary to prepare liter quantities of specially constructed media. Furthermore, the probability of contamination increases during such long-term experiments since the culture dishes must be handled commensurately more times for medium changes. A method was sought that would reduce the duration of nutritional experiments, the quantity of media prepared, and still offer results and reproducibility comparable to existing methods for monolayer cell cultures. This method is a modification of Falcon (60 x 15 mm) plastic culture dishes commonly used for the growth of monolayer cell cultures.

MATERIALS AND METHODS

A brass cylinder 41.5 mm x 35 mm x 2.5 mm was brazed on center to a 2 mm thick circular brass plate with a diameter of 50 mm. Holes were drilled in the plate to accommodate the stems of brass brads with a length of 12.6 mm and with heads approximately 2 mm in diameter. A total of 144 evenly spaced holes were drilled in five concentric circles with the outer circle having a radius of 16.9 mm. The brads were placed in the holes leaving the heads exposed. The cylinder with attached plate and brads was inverted leaving the remaining opening of the cylinder uppermost. A Lenk, Model 675, 1 watt, 115 V soldering iron (Boston, Mass.) was placed to center into the cylinder and the void filled with molten lead (Fig. 1). All materials were available at a local plumbing supply or hardware store. A rheostat was used to maintain a temperature adequate to soften the plastic and to permit imprinting of the brass heads into the bottom dishes. A clamp mounted on a laboratory stand was used to support the device. Mineral oil was coated lightly over the surface of the plate and the exposed heads of the brads to prevent adhesion to the plastic culture dish during imprinting. Pressure to facilitate impressing was exerted with a No. 12 neoprene rubber stopper. Care was exercised to avoid twisting of the culture dish during this process. After imprinting, the culture dish was washed with Alconox to remove the mineral oil, copiously rinsed with glass distilled water and dried. The bottom of a 35 x 10 mm Falcon culture dish was removed next by abrasion with a flat bastard file around the periphery, and the edge of the resulting plastic cylinder smoothed on 000 emery cloth. This plastic cylinder was placed within the 60 x 15 mm culture dish so as to enclose the imprinted wells and fixed to the culture dish with No. 40-8 acrylic cement (G-C Electronics Co., Los Angeles, Calif.). It is important that the

Fig. 1. Imprinting Device.
A. Brass plate 50 mm. x 2 mm.
B. Brass brads.
C. Brass cylinder 41.5 mm. x 35 mm. x 2.5 mm.
D. Soldering iron.

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two concentric chambers are completely sealed from each other. The culture dish was washed again and rinsed as previously described. Sterilization was accomplished either by ultraviolet irradiation (dose of 20 ergs per sq. mm per sec.) for two hours or by exposure to ethylene oxide for 30 minutes. Mouse "L" cells and Y-1 adrenal cells were obtained from the American Type Culture Collection Cell Repository, Rockville, Maryland.

All cultures were grown in a basal medium composed of Ham's F10 and supplemented with 15% horse serum and 2.5% fetal calf serum.[3] All media were constructed from the best commercially available chemicals. Sera were obtained from Grand Island Biological Co., Grand Island, N. Y. Sterilization of media was by Millipore filtration and storage was at -20° C. Incubation of the cultures was in a water-jacketed incubator with a water saturated atmosphere of 95% air and 5% CO_2. The near 100% humidity of the incubation chamber was insured by connecting several ¼ x 10 inch stone fish tank bubblers to the gas mixture supply and placing them in the water chamber used to humidify the atmosphere of the incubator. Stock cultures of mouse "L" cells were dissociated with 0.1% trypsin in a phosphate buffered saline solution. Stock monolayer cell cultures of Y-1 mouse adrenal cortex tumor cells were dispersed using 10^{-3}M EGTA in a Krebs phosphate buffer solution minus calcium and magnesium ions. Cell number estimations were made with a hemocytometer counting chamber.

Aliquots of a single cell suspension containing about 500-1000 cells per ml were placed in the center chamber containing the imprinted wells. Sterile glass distilled water (2 ml) was placed in the outer well and served as a hydration chamber. The culture dish was incubated for 1-2 hours in order to permit cell attachment. The growth medium was aspirated and a gentle rinse with medium carried out to remove unattached cells. The medium was removed further by carefully tipping the dish to one side and aspirating the contents of the inner chamber. With this procedure, medium was retained in the wells, leaving the adjacent flat surfaces nearly dry. The culture dish was returned then to the incubator. Medium changes consisted of adding 1 ml of growth medium to each center chamber with the imprinted wells, followed by aspiration as described. Medium changes were made as noted in the text.

Control monolayer cell cultures of both established lines were used to determine the mean population doubling time. These were carried out using an initial cell population of 1 - 2.5 x 10^5 cells and 3 ml of the growth medium in unmodified Falcon plastic 60 x 15 mm culture dishes. The spent medium was replaced every three days with 3 ml of fresh medium. The population doubling time was calculated by estimating either the cell number or total cellular protein in at least duplicate cultures during the period of exponential growth. The methods used for determining cell number and total cellular protein have been described previously.[4]

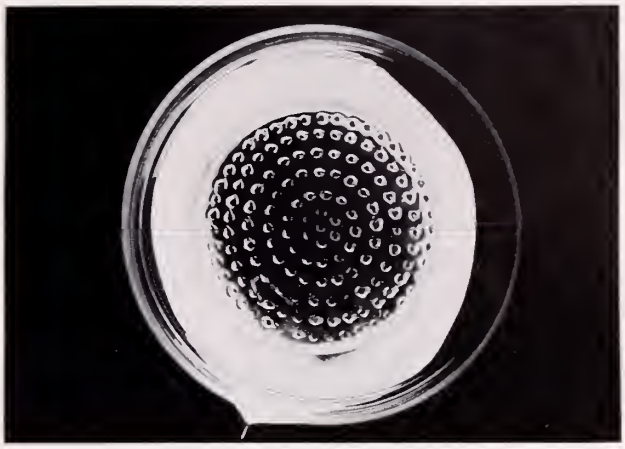

Fig. 2. A 65 x 15 mm. Falcon plastic culture dish imprinted with 144 wells.

Results

Fig. 2 is a photograph of a culture dish constructed as described in "Materials and Methods." It can be seen that the wells are not symmetrically round and uniform in size. This is the result of slight variations in the size and shape of the heads of the brads which were available commercially. The measurement of ten such heads was made with a Vernier millimeter caliper which permitted measurements to 0.1 mm. The mean diameter obtained was 1.5 mm $\pm$ 0.2 mm. Assuming the heads to be perfect half-spheres, this calculates to a mean volume of 0.9 ul $\pm$ .3 ul per well. These should be considered approximate values. The method used to imprint the wells did not destroy the optical properties of the plastic or impair the attachment of cells.

Eight hours after plating, the wells were examined. The cells were counted in randomly selected wells by direct observation with an inverted microscope using phase contrast (100x). Usually not more than 10 wells per plate with 1-20 cells per well were chosen for continued observation. Cells attached to the flat surfaces of the culture dish between the wells degenerated rapidly due to the reduced amount of growth medium available to them following aspiration.

The volume of growth medium retained in the wells after aspiration proved to be adequate for the growth of the contained cells. Table 1 illustrates the growth of varying numbers of Y-1 cells which occurred in seven wells. Fewer than 4 Y-1

GROWTH PERIOD (hours)	Cells per Well Plated	24	45.5	83.5	141.5	186
#1	2	2	2	0	-	-
#2	4	2	1	0	-	-
#3	5	5	5	8	27	60*
#4	3	3	1	0	-	-
#5	4	4	3	5	15	38
#6	3	2	1	3	0	-
#7	22	20	23	40	116*	251*

*These values represent the average of at least 5 separate counts

Table 1. The increase in the cell number of Y-1 mouse adrenal cortex tumor cells observed in seven separate wells (No. 1-No. 7) present in one Falcon culture dish modified as described in the text.

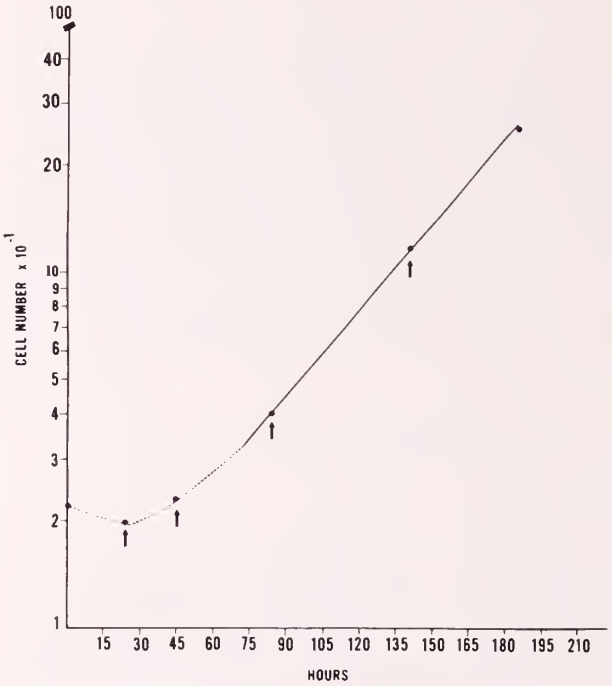

Fig. 3. Exponential growth of Y-1 cells in a well. Growth medium Ham's F10 supplemented with 15% horse serum and 2.5% fetal calf serum. Population doubling time is estimated to be about 36 hours.
↑ Indicates medium changes as described in text.

cells per well do not appear to survive. It was observed that a single "L" cell in a single well was more likely to proliferate than a single Y-1 cell in a separate well. Possible reasons for these observations will be proposed in the discussion. A plot of the data from well No. 7 appears in Fig. 3. The population doubling time as estimated during exponential growth is about 36 hours. This favorably agrees with the control value of 33 hours and with previous observations.[5] More frequent changes of media were necessary to maintain the rate of exponential growth as compared to control cultures grown under the conditions described in "Materials and Methods." A similar experiment carried out with mouse "L" cells using the same basal medium and serum supplementation is presented in Fig. 4. The population doubling time of control "L" cell cultures was 26 hours with the same growth medium. In order to maintain a nearly similar population doubling time with "L" cells growing in medium limited to wells, it was necessary to change the medium even more frequently than that of the Y-1 culture. The gradual onset of the stationary phase of growth is apparent as the cell population density reaches the limit imposed by the volume of growth medium contained in the well.

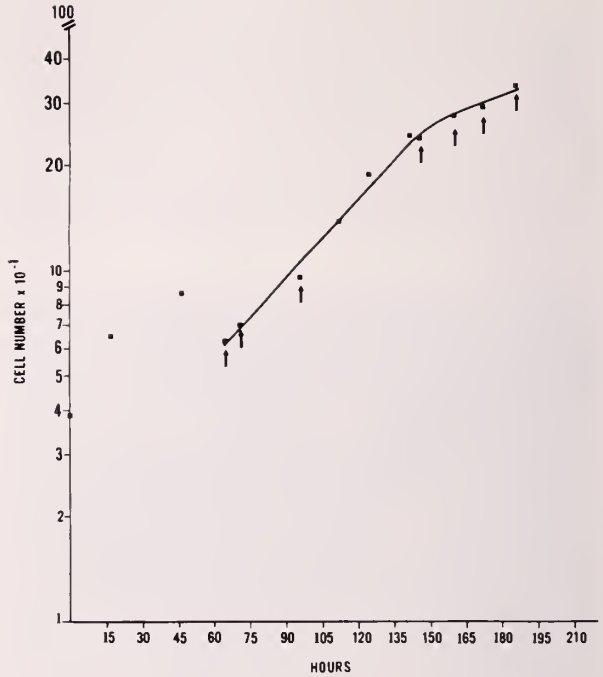

Fig. 4. Exponential growth of mouse "L" cells in a well. Growth medium Ham's F10 supplemented with 15% horse serum and 2.5% fetal calf serum. Population doubling time is estimated to be approximately 31 hours.
↑ Indicates fresh medium changes which were made according to the method described in the text. Changes were made either when there was a loss in the cell number or when the increase in cell number was less than that expected from an extrapolation of the previous determinations of cell number.

Experiments usually were terminated because of the difficulty encountered in not being able to accurately distinguish individual cells in a population that exceeded 250 cells in a well.

Discussion

The data presented show that small numbers of cells (< 10) can proliferate in small volumes of media providing dehydration is prevented or greatly reduced during incubation. It is believed that the provision of a hydration chamber with a surface area of nearly 60% of the total surface area present in the bottom plate of a Falcon 60 x 15 mm culture dish which has a measured diameter of 52 mm serves this purpose adequately. The construction of shallow wells is preferred to the inverted cylindrical cones offered by Falcon in their Microtest plates, No. 3034. The primary objection to the wells of the Microtest plate is that cells do grow on the sides, making detection and enumeration nearly impossible (Fig. 5).

The volume of growth medium may offer too large a gradient for the number of cells present to create a favorable extracellular concentration if a basal growth medium does not have optimum concentrations of the nutrients necessary for growth. Eagle[6,7] has demonstrated this relationship especially in respect to the so-called "non-essential" amino acids. This may account for the inability to grow fewer than four Y-1 cells in the wells described. Pierson and Temin[8] have reported recently an insulin-like growth factor with multiplication-stimulating activity (MSA) which they isolated from calf serum. Temin[9] has noted further that chicken fibroblasts can synthesize MSA. The ratio of cell number to volume of growth medium would be a critical factor if Y-1 cells synthesized MSA. The viability of cells upon subculture varies and can contribute to the observation that all single cells do not proliferate in separate wells.

Small half-sphere wells of various diameters in a subculture dish can offer the advantage of extremely small volumes for the direct observation of the growth of cells. It can be calculated that a Falcon 60 x 15 mm culture dish, when filled with 3 ml of growth medium has a cylindrical volume of 8.8 ul for every circular flat surface measuring 2 mm in diameter. A well, constituting a half-sphere with a diameter of 2 mm, has an internal surface area of approximately 6.28 sq. mm with a volume of 2.1 ul. Reduction of the diameter of the half-sphere to 1.2 mm further reduces this volume to 0.45 ul (Table 2). Therefore, although the surface area available for monolayer cell growth within a half-sphere of any given diameter is double that of a flat surface with the same diameter, the evaporation area of the liquid-atmosphere interface is the same.

It would be possible with accurate machining to construct a culture dish with half-spheres arranged concentrically with gradually diminishing volumes for each circle of wells. This would facilitate studies relating the dependency of cell population density to micro amounts of biologically active material and the selection of variant strains. Clones originating from single cells can be established with this procedure and subcultured using the ring cloning technique.[10] Culture dishes so constructed might also prove most useful to investigators who wish to observe single cell interactions involving cell hybridization[11] or immunological responses.[12]

Fig. 5. Photomicrograph demonstrating the attachment and growth of L cells on the lateral well surface of a Falcon 3034 Microtest plate. x100 (Cells from a single cell suspension readily attach to the lateral surface and often are not detected in the same focal plane as cells growing on the bottom surface. This difficulty is not encountered in the shallow well described as the entire surface for growth lies within the field of observation and most of this surface is within the focal plane.)

Diameters	A Surface area of ½ Sphere ($4\pi r^2/2$)	B Cylindrical Volume of 3ml Growth Medium with Same Surface Area Indicated in Column A.	C Volume of ½ Sphere ($4/3\ \pi r^3/2$)
2.0 mm	6.28 mm²	8.8 μl*	2.1 μl
1.2 mm	2.26 mm²	3.2 μl*	0.45 μl

* Volume calculated as area x height of medium. Height of 3 ml of medium in a Falcon 60 x 15mm culture dish was estimated as 1.4mm (diameter of bottom dish measured 52mm).

Table 2. The volumes of ½ spheres at two different diameters compared to the volumes of cylinders with similar surface areas.

Summary

Exponential growth of two established cell lines was observed using small cell numbers in a minute volume of growth medium by constructing small shallow wells in plastic culture dishes. The restriction of growth medium to these wells and the prevention of excessive dehydration facilitates the direct observation of the multiplication of single cells. The establishment of 144 wells per culture

Continued on Page 151

Papillary Squamous Cell Carcinoma of the Limbus

A Case Report

MERRILL J. KING, JR., M.D.*

While malignant tumors of the conjunctiva are rather rare, the limbus of the eye can be host to a wide and confusing variety of other lesions.[1,2] These include benign processes such as pinguecula, pterygium, Bitot's spot of xerosis, lymphangiectasia, blood vessel telangiectases, and others. A pseudopterygium may follow injuries to this region as may conjunctival inclusion cysts or keloids. Dermoids may occur at the limbus. Nevi are common and, as elsewhere, occasionally may become malignant.

Caterpillar hairs imbedded in the conjunctiva may produce ophthalmia nodosa. Several years ago the author and his father examined a patient with what appeared to be an epithelial tumor of the limbus. Examination of the excised mass showed chronic inflammatory changes surrounding the larva of a gnat which had flown into the eye and oviposited into the conjunctiva before drowning in the patient's tears!

Numerous authors have described epithelial neoplastic changes ranging from epidermalization and leukoplakic metaplasia through Bowen's type lesions and carcinoma-in-situ to frank squamous cell carcinomas.[3,4,5,6] True adenomas are very rare.[7]

Distinguishing one from another, the various lumps and bumps in this wide array can be very perplexing. This is especially so for the nonophthalmologist who usually has the added disadvantage of having no slit-lamp biomicroscope at hand.

Fortunately even the true carcinomas tend to grow slowly in the limbal region. Because they usually occur in the interpalpebral fissure area, they are not apt to be overlooked. Deep invasion of the eyeball or posterior spread toward the fornices is not common or it occurs quite late. Bowman's membrane of the cornea is usually an effective barrier against deep penetration even though there may be considerable growth out over the cornea. Metastases can occur but they are generally late and rare.

Most present day authors advise simple local excision using a lamellar keratectomy to deal with any extension of the tumor onto the corneal surface.

Fig. 1

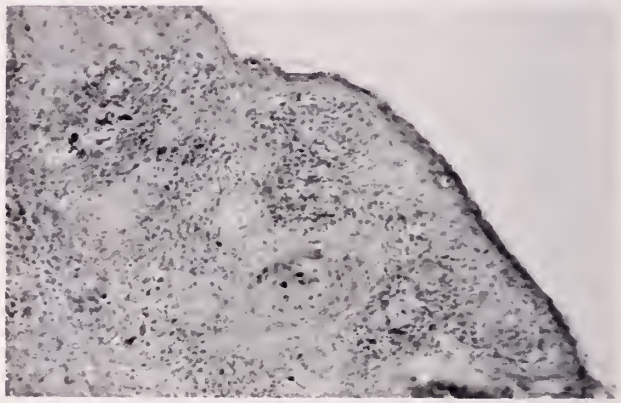

Fig. 2

Beta radiation from a ^{90}Sr source, or other contact radiation techniques, can be used in conjunction with surgery or to treat recurrences.[8,9] Enucleation is seldom advocated today unless there is obvious intraocular penetration.

CASE REPORT

A 56-year-old white male lumberjack was referred to the Veterans Administration Center, Togus, by his local physician who had made a tentative diagnosis of pterygium of the right eye. Six months previously the patient had noted a mass on the conjunctiva just nasal to the limbus. Subsequent growth was slow but steady. There were no symptoms except for mild irritation of the eye.

Vision was better than 20/25 in each eye. In the right eye a pink,

*Chief, Eye Section, Surgical Service, Veterans Administration Center, Togus, Maine 04330.

circular mass about 4.0 mm in diameter was located on the conjunctiva just below the horizontal meridian at the limbus on the nasal side. It was slightly elevated above the surrounding conjunctiva. Biomicroscopy showed the surface to consist of numerous fine papillae, each with a central vascular core. The conjunctival vessels supplying the tumor were enlarged (see Fig. 1). Corneal invasion extended only 1.0 mm from the limbus and was very superficial. Except at its limbal edge, the tumor was freely movable over the underlying sclera. There were no signs of intraocular invasion. The intraocular tension was normal (15 mm Hg) in each eye. Except for retinal arteriolar narrowing consistent with a brachial blood pressure of 210/125, no other ocular abnormalities were noted.

After regulation of the hypertension, the tumor was excised under local infiltration anesthesia. A lamellar excision of the affected cornea and adjacent sclera was combined en bloc with a wedge resection of the conjunctiva surrounding the tumor. Microscopic sections showed a papillary squamous cell carcinoma. Prickle cells and epithelial pearls were apparent (see Fig. 2).

Healing was uneventful. Follow-up examination showed no recurrence two months postoperatively. Continued observation is planned.

Comment

Most limbal tumors occur near the horizontal meridian, where lid protection is the least effective. By way of partial compensation, the tumors are easily visible when they do occur and frequently attract the early attention of the patient or spouse if not that of the examining physician. The exposed location of the tumors suggest that prolonged irritation from wind, sunlight, and dust may in part be responsible for their genesis. This patient, a woodsman, was exposed for years to all of these agents. (He also had several basal cell and squamous cell carcinomas removed from his face and hands during the previous few years.)

With rare exceptions, conservative excision with or without added radiation will save the eye as well as the patient. As Ash[3] has said, "If one must have a cancer, the limbus would be as safe a place as any to have it."

References

1. Reese, A. B.: Tumors of the eye. New York: Harper & Bros., 1951.
2. Hogan, M. J., and Zimmerman, L. E.: Ophthalmic pathology, ed. 2. Philadelphia: W. B. Saunders Co., 1962.
3. Ash, J. E., and Wilder, H. C.: Epithelial tumors of the limbus. Am. J. Ophthalmol. 25: 926-932, Aug., 1942.
4. Carroll, J. M., and Kuwabara, T.: A classification of limbal epitheliomas. Arch. Ophthalmol. 73: 545-551, Apr. 1965.
5. McGavic, J. S.: Intraepithelial epithelioma of the cornea and conjunctiva (Bowen's disease). Am. J. Ophthalmol. 25: 167-176, Feb., 1942.
6. Kennedy, R. J.: Epithelial plaques of the conjunctiva. Am. J. Ophthalmol. 35: 843-847, June, 1952.
7. Francois, J.: Adenoma of the limbal conjunctiva. Br. J. Ophthalmol. 35: 237-241, Apr., 1951.
8. Paufique, L.: Physiothérapie des épithéliomas du limbe. Bull. Soc. Ophthalmol. Fr. 69: 478-483, Apr., 1969.
9. Papillon, J.: La radiothérapie de contact des épithéliomas due limbe scléro-cornéen. Ann. Radiol. (Paris) 14: 609-616, Jul.-Aug., 1971.

MODIFICATION OF PLASTIC CULTURE DISHES FOR SINGLE CELL OBSERVATIONS

Continued from Page 149

dish not only greatly reduces the total number of culture dishes required but also the quantity of media that is necessary to prepare. Restriction of cells to the wells limits any possible migration. The reduced volume of growth medium in each well results in reaching the stationary phase of logarithmic growth in a shorter period of time. Frequent changes of medium were required in order to obtain mean population doubling times comparable to controls with considerably larger volumes of growth medium and larger initial cell populations. The advantages of this method include the requirement for considerably smaller volumes of media and fewer culture dishes since direct enumeration of cells can be made. It is considered that improvements in the construction of the wells can enhance the usefulness of this method and suggestions have been made accordingly.

References

1. Moscona, A., Trowell, O. A., and Willmer, E. N.: Methods. In: Cells and Tissues in Culture. Methods, Biology and Physiology, E. N. Willmer (ed.), Vol. 1. London: Academic Press, pp. 60-70, 1965.
2. Eagle, H.: The specific amino-acid requirement of a mammalian cell (Strain L) in tissue culture. J. Biol. Chem. 214: 839-853, 1955.
3. Ham, R. G.: An improved nutrient solution for diploid Chinese hamster and human cell lines. Exp. Cell Res. 29: 515-526, 1963.
4. Cuprak, L. J. and Sato, G.: The nutritional requirements of mouse adrenal cortex tumor cells in culture. Exp. Cell Res. 52: 632-645, 1968.
5. Sato, G. H. and Yasamura, Y.: Retention of differentiated function in dispersed cell culture. Trans. N. Y. Acad. Sci., Ser. II, 28: 1063-1079, 1966.
6. Eagle, H. and Piez, K.: The population-dependent requirement by cultured mammalian cells for metabolites which they can synthesize. J. Exp. Med. 116: 29-43, 1962.
7. Eagle, H., Washington, C. L., Levy, M., and Cohen, L.: The population-dependent requirement by cultured mammalian cells for metabolites which they can synthesize. II. Glutamic acid and glutamine; aspartic acid and asparagine. J. Biol. Chem. 241: 4994-4999, 1966.
8. Pierson, R. W., Jr., and Temin, H. M.: The partial purification from calf serum of a fraction with multiplication-stimulating activity for chicken fibroblasts in cell culture and with non-suppressible insulin-like activity. J. Cell. Physiol. 79: 319-329, 1972.
9. Temin, H. M.: Stimulation by serum of multiplication of stationary chicken cells. J. Cell. Physiol. 78: 161-170, 1971.
10. Puck, T. T., Marcus, P. I., and Cieciura, S. J.: Clonal growth of mammalian cells in vitro. J. Exp. Med. 103: 273-284, 1956.
11. Harris, H., Sidebottom, E., Grace, D. M., and Bramwell, M. E.: The expression of genetic information: A study with hybrid animal cells. J. Cell Sci. 4: 499-525, 1969.
12. Hellstrom, I.: A colony inhibition (CI) technique for demonstration of tumor cell destruction by lymphoid cells in vitro. Int. J. Cancer 2: 65-68, 1967.

—News From Blue Cross and Blue Shield—

REGIONAL BLOOD PROGRAM GIVEN SUPPORT

Richard F. Nellson, President of Maine Blue Cross and Blue Shield, and James H. Harvey, Jr., Director of Regional Blood Banks, Inc., have signed an administrative agreement which will allow Maine Blue Cross and Blue Shield to offer the blood program to subscribers and will give Regional Blood Banks, Inc. administrative support from the Blue Cross organization.

Mr. Nellson stated at the signing: "We have supported the Regional Blood Bank concept since its inception over four years ago, and we consider the program a necessity for the State of Maine. We will do everything in our power to stimulate the growth in membership of Regional Blood Banks, and we encourage every healthy Maine citizen to give at least one pint of blood a year to insure that no one will ever lose his life for want of blood in Maine."

The Regional Blood Bank system offers a centralized source of good, free blood and ready access to all types of blood. Before the blood bank system was available in Maine, hospitals had to draw and process their own blood, and coordination of hospitals to obtain critically needed blood was difficult. Regional Blood Banks, operating with a system almost identical to the Red Cross Blood Bank concept, has the personnel, expertise and equipment to handle the needs of all patients in member hospitals.

Regional Blood Banks, Inc., a certified member of the American Association of Blood Banks, has the endorsement of all its contracting hospitals, as well as the many pathologists who have been associated with the program. The Regional Blood Banks, of which there are now three located in Portland, Auburn and Waterville, offer good, free blood to 26 contracting hospitals and hope to expand to 42 hospitals throughout Maine in the near future.

"Blue Cross and Blue Shield members who give blood through the Regional program will incur absolutely no expenses for blood should they require it. The blood is free, and the administrative charge is absorbed by their Blue Cross coverage," Nellson stated. "Our support of this program is indicative of our objective to provide benefits for the best possible healthcare at the lowest possible cost to the consumer. We are striving to encourage centralization of healthcare facilities wherever possible, and the Regional Blood Bank system is a major step in that direction."

Detailed information about the program can be obtained from Regional Blood Banks, Inc., 966 Riverside Street, Portland, Maine 04101.

MITSIE Provides Emergency Medical Service For Infants

Operation MITSIE — a new emergency medical service — is now available to Maine families. MITSIE (Maine Incubator Transfer Service for Infant Emergencies) is a co-operative joint emergency medical service of the Maine Army National Guard, State Police, and the Northern Maine Chapter of the March of Dimes.

Realizing the need for immediate transfer of ailing newborn infants to a major medical facility for emergency treatment, Operation MITSIE has been organized and is ready for utilization by the public — on a physician's direct request. One portable incubator has been purchased with March of Dimes funds. It will be located at the Dow Field Operations Center of the 112th Medical Company (Air Ambulance) of the Maine Army National Guard, the unit providing the emergency air evacuation.

MITSIE is a true isolation-type, intensive care transport incubator. It is a self-contained unit and fully equipped with all life support systems for maintaining infant body temperature and a properly humidified and oxygenated atmosphere. MITSIE offers continuous observation and immediate access for treatment to the infant.

The Maine State Police will act as the releasing agent and the Maine Army National Guard will provide emergency air transportation when the condition of the child makes commercial transportation inadequate.

Both State Police and Army National Guard medical corpsmen are being trained in the care and operation of MITSIE, but a medical person must accompany every ill infant to attend its care and medical needs.

To obtain the use of MITSIE, the physician attending the infant must declare that the infant is in need of emergency transportation and notify the nearest barracks of the Maine State Police.

The use of MITSIE and the services of the State Police and the Maine Army National Guard are available throughout the State of Maine at no cost to the family of the infant, but the costs of private commercial transportation used to transfer MITSIE to or from airport, etc., must be paid by the child's family.

This information was obtained from the Maine Army National Guard. All questions about the service should be sent to the Northern Maine Chapter, March of Dimes, Post Office Box 22, Brewer, Maine. The office telephone number is 989-3376.

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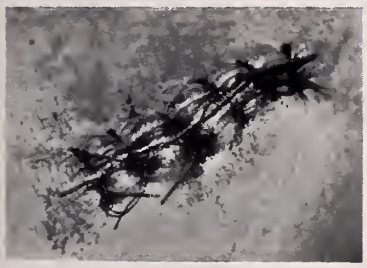

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Contraindications: Glaucoma, obstructive disease of the gastrointestinal tract, obstructive uropathy, intestinal atony, toxic megacolon, hiatal hernia associated with reflux esophagitis, or unstable cardiovascular adjustment in acute hemorrhage.

Warnings: Patients with severe cardiac disease should be given this medication with caution.

Fever and possibly heat stroke may occur due to anhidrosis.

In theory a curare-like action may occur, with loss of voluntary muscle control. For such patients prompt and continuing artificial respiration should be applied until the drug effect has been exhausted.

Diarrhea in an ileostomy patient may indicate obstruction, and this possibility should be considered before administering Pro-Banthīne.

Precautions: Since varying degrees of urinary hesitancy may be evidenced by elderly males with prostatic hypertrophy, such patients should be advised to micturate at the time of taking the medication.

Overdosage should be avoided in patients severely ill with ulcerative colitis.

Adverse Reactions: Varying degrees of drying of salivary secretions may occur as well as mydriasis and blurred vision. In addition the following adverse reactions have been reported: nervousness, drowsiness, dizziness, insomnia, headache, loss of the sense of taste, nausea, vomiting, constipation, impotence and allergic dermatitis.

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The contraindications and precautions applicable to Pro-Banthīne 15 mg. should be observed.

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Contraindications: Pre-existing elevated serum potassium. Hypersensitivity to either component. Continued use in progressive renal or hepatic dysfunction or developing hyperkalemia.

Warnings: Do not use dietary potassium supplements or potassium salts unless hypokalemia develops or dietary potassium intake is markedly impaired. Enteric-coated potassium salts may cause small bowel stenosis with or without ulceration. Hyperkalemia (> 5.4 mEq/L) has been reported in 4% of patients under 60 years, in 12% of patients over 60 years, and in less than 8% of patients overall. Rarely, cases have been associated with cardiac irregularities. Accordingly, check serum potassium during therapy, particularly in patients with suspected or confirmed renal insufficiency (e.g., elderly or diabetics). If hyperkalemia develops, substitute a thiazide alone. If spironolactone is used concomitantly with 'Dyazide', check serum potassium frequently—both can cause potassium retention and sometimes hyperkalemia. Two deaths have been reported in patients on such combined therapy (in one, recommended dosage was exceeded; in the other, serum electrolytes were not properly monitored). Observe patients on 'Dyazide' regularly for possible blood dyscrasias, liver damage or other idiosyncratic reactions. Blood dyscrasias have been reported in patients receiving Dyrenium (triamterene, SK&F). Rarely, leukopenia, thrombocytopenia, agranulocytosis, and aplastic anemia have been reported with the thiazides. Watch for signs of impending coma in acutely ill cirrhotics. Thiazides are reported to cross the placental barrier and appear in breast milk. This may result in fetal or neonatal hyperbilirubinemia, thrombocytopenia, altered carbohydrate metabolism and possibly other adverse reactions that have occurred in the adult. When used during pregnancy or in women who might bear children, weigh potential benefits against possible hazards to fetus.

Precautions: Do periodic serum electrolyte and BUN determinations. Do periodic hematologic studies in cirrhotics with splenomegaly. Antihypertensive effects may be enhanced in postsympathectomy patients. The following may occur: hyperuricemia and gout, reversible nitrogen retention, decreasing alkali reserve with possible metabolic acidosis, hyperglycemia and glycosuria (diabetic insulin requirements may be altered), digitalis intoxication (in hypokalemia). Use cautiously in surgical patients. Concomitant use with antihypertensive agents may result in an additive hypotensive effect.

Adverse Reactions: Muscle cramps, weakness, dizziness, headache, dry mouth; anaphylaxis; rash, urticaria, photosensitivity, purpura, other dermatological conditions; nausea and vomiting (may indicate electrolyte imbalance), diarrhea, constipation, other gastrointestinal disturbances. Rarely, necrotizing vasculitis, paresthesias, icterus, pancreatitis, and xanthopsia have occurred with thiazides alone.

Supplied: Bottles of 100 capsules.

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Honorary Pin Recipients Receive Awards at 1973 Annual Session of the M.M.A.

Presentation of the Association's Honorary Pins were made by George W. Wood, III, M.D., President of the M.M.A., at the Annual Banquet, Monday evening, June 18 at 7:00 P.M.

FIFTY-YEAR PINS

Fifty-Year Lapel Pins will be presented to the following members who were graduated from Medical School in 1923:

Androscoggin County

CHARLES F. BRANCH, M.D. – Dr. Branch was President of the Maine Medical Association in 1969-1970 and President-elect in 1968-1969. He was Councilor for the Second District from 1963 to 1968, the last year as Council Chairman. A native of Amherst, Massachusetts, Dr. Branch was graduated from St. Johnsbury Academy in Vermont in 1917, received his medical degree from the University of Vermont College of Medicine in 1923 and served a residency in Pathology at the Boston City Hospital from 1923 to 1925. Dr. Branch taught at Jefferson Medical College in Philadelphia from 1925 to 1926 and at Boston University from 1926 to 1946. He was professor of Pathology at Boston University from 1932 to 1946, dean of the medical school from 1943 to 1946 and was associate director of Evans Memorial for Clinical Research from 1939 to 1944. He was licensed as a specialist by the American Board of Pathologists in 1937. From 1946 to 1947, he was executive director of the Children's Medical Center in Boston and from 1947 to 1950, assistant director of the American College of Surgeons. In 1950, Dr. Branch was appointed director of Laboratories and Pathologist-in-Chief at the Central Maine General Hospital in Lewiston, Maine. He has served as Consulting Pathologist at the Rumford Hospital, Franklin County Memorial Hospital, Goodall Hospital and Stephens Memorial Hospital and as medical examiner for Androscoggin County. He is also one of the founders of the Maine Pathological Society and the Maine Medico-Legal Society, of which he was president from 1961 to 1963. Dr. Branch was appointed Maine's first Chief Medical Examiner in May 1968. This position was created by the legislature in special session in January 1968 to give the State some central direction and supervision in the medical-legal aspects of homicides and unattended or unexplained deaths.

Cumberland County

ABRAM I. COHEN, M.D. – Born in Boston, Dr. Cohen attended Harvard University and received his medical degree from Tufts University School of Medicine in 1923. He interned at the Mt. Sinai Hospital in Philadelphia from 1923 to 1924 and took a postgraduate course at the University of Chicago. Dr. Cohen practiced in the Boston area where he was affiliated with the New England Medical Center, the Beth Israel Hospital, the Forsyth Dental Infirmary and was an instructor in E.N.T. at Tufts University School of Medicine. He located in Harrison, Maine in 1953 and was formerly chief of staff of the Northern Cumberland Memorial Hospital in Bridgton, Maine.

EUGENE E. O'DONNELL, M.D. – Dr. O'Donnell was President of the Maine Medical Association in 1958-1959, President-elect in 1957-1958, Council Chairman in 1956-1957 and, prior to that, had served as Councilor for the First District. Born in Lubec, Maine, Dr. O'Donnell attended Bates College, was graduated from Bowdoin College and received his medical degree from Yale University School of Medicine in 1923. He did postgraduate work at the Maine General Hospital and at the New Haven Hospital. Dr. O'Donnell has practiced General Surgery in Portland since 1924 and resides in Cape Elizabeth, Maine.

THEODORE M. STEVENS, M.D. – Dr. Stevens, a native of Portland, Maine, has practiced in that city since 1925. He was graduated from the University of Maine and received his medical degree from Harvard Medical School in 1923. Dr. Stevens interned at the St. Mary's Hospital in Dorchester, Massachusetts and the City Hospital in Worcester, Massachusetts. Specializing in Obstetrics and Gynecology, Dr. Stevens has been affiliated with the Maine General Hospital, the Maine Eye and Ear Infirmary, the Maine Medical Center and the Mercy Hospital.

Kennebec County

HOWARD F. HILL, M.D. – Dr. Hill was graduated from Colby College and received his medical degree from Harvard Medical School in 1923. He interned at the Chestnut Hill Hospital in Philadelphia, served a residency at the Wills Eye Hospital in Philadelphia from 1924 to 1926 and took a postgraduate course in Vienna, Austria. Now semi-retired, Dr. Hill has practiced Ophthalmology in the Waterville area since 1925,

Dr. Branch

Dr. Cohen

Dr. Stevens

Dr. Hill

where he has been affiliated with the Thayer Hospital, the Veterans Administration Center, the Rockland General Hospital and the Gardiner General Hospital. He taught cataract surgery at the Academy of Ophthalmology for 12 years, lectured in 16 countries and has written many publications.

Knox County

BARBARA B. STIMSON, M.D. – Born in New York City, Dr. Stimson was graduated from Vassar College in 1919 and received her medical degree from the Columbia University College of Physicians and Surgeons in 1923. She interned and served a Surgical Fellowship at the Presbyterian Hospital in New York City. Following this, Dr. Stimson was a post-doctoral Fellow at Western Reserve University and Columbia University. Her hospital appointments include the Presbyterian Hospital, the Vassar Brothers Hospital and St. Francis Hospital in Poughkeepsie, New York. During World War II, she served as a Major in the Royal Army Medical Corps, British Army. Dr. Stimson located in Owl's Head, Maine in 1963, where she now resides.

Oxford County

CHESLEY W. NELSON, M.D. – Dr. Nelson, born in Southport, Maine, was graduated from Bates College in 1909 and received his medical degree from the University of Vermont College of Medicine in 1923. He interned at the Mary Fletcher Hospital in Burlington, Vermont. Dr. Nelson, a General Practitioner, has practiced in Norway, Maine since 1924 and has been on the staff of the Stephens Memorial Hospital since 1957.

York County

HERMAN G. SANDVOSS, M.D. – A native of Fuerth, Germany, Dr. Sandvoss received his medical degree from the University of Munich in 1923, and interned and served a surgical residency at the University of Munich. He did postgraduate work relating to Occupational Medicine at the Menninger Foundation. Specializing in Industrial Medicine, Dr. Sandvoss was Medical Director and Chief Surgeon, Standard Oil Co. of New Jersey, 1928-32, full time; Medical Director and Chief Surgeon, Aruba, N.W.I., 1932-41, full time; Medical Department, Rockefeller Center, Inc., New York, 1942-46, part time; Medical Director, Armour & Co., New York area, 1946-56, part time; Assistant Medical Director, Daily News Syndicate, New York, 1951-60, part time; Medical Director, The Federal Reserve Bank of New York, 1960-69, full time; and from 1970 to the present, in charge of the Medical Department, American Cyanamid, Sanford, Maine, part time. Since 1969, Dr. Sandvoss has practiced in Kennebunkport where he now resides.

FIFTY-FIVE-YEAR PINS

Fifty-Five-Year Pins will be presented to the following members who received Fifty-Year Pins in 1968:

Cumberland County

WILLIAM E. FREEMAN, M.D. – Born in West Lynn, Massachusetts, Dr. Freeman attended Bowdoin College and received his medical degree from Bowdoin Medical School in 1918. He interned and served a residency at the Lawrence General Hospital in South Lawrence, Massachusetts and took a postgraduate course at the New York Skin and Cancer Hospital. Specializing in Dermatology, Dr. Freeman practiced in Standish, Maine and then located in Yarmouth, Maine in 1927, where he now resides. His hospital appointments include the State Street Hospital and the Maine Eye and Ear Infirmary in Portland.

Knox County

HARRY A. NAUMER, M.D. – Dr. Naumer was graduated from Columbia University in 1915 and received his medical degree from the Columbia University College of Physicians and Surgeons in 1918. He interned at the Brooklyn Hospital, served a residency at the Babies Hospital in New York City from 1919 to 1920 and did postgraduate work at the Children's Hospital in Boston. Specializing in Pediatrics, his hospital appointments in New York included the Greenpoint Hospital, the Brooklyn Hospital and the Long Island College Hospital. In the 1950's, Dr. Naumer located in Port Clyde, Maine where he was affiliated with the St. George Child Health Clinic until 1968. From 1916 to 1920, he served as a 1st Lieutenant in the Medical Reserve Corps. Dr. Naumer is a native of New York.

Penobscot County

W. MERRITT EMERSON, M.D. – Dr. Emerson attended the University of Vermont and received his medical degree from the University of Vermont College of Medicine in 1918. He interned at the Mary Fletcher and Fanny Allen Hospitals in Burlington, Vermont and the New York Lying-In Hospital. Dr. Emerson, who practices Cardiology, located in Bangor in 1918, where he has been affiliated with the St. Joseph Hospital. He is a native of Carmel, Maine.

Piscataquis County

CHARLES N. STANHOPE, M.D. – A native of Charleston, Maine, Dr. Stanhope now resides in Dover-Foxcroft. He was graduated from Bates College in 1912 and received his medical degree from Bowdoin Medical School in 1918. From January

Dr. Nelson

Dr. Naumer

Dr. Emerson

Dr. Stanhope

1918 to August 1918, he served as Assistant Superintendent of the Western Maine Sanatorium and then located in Dover-Foxcroft where he was a staff member of the Mayo Memorial Hospital. Dr. Stanhope specialized in the field of Public Health and was affiliated with the State Department of Health from 1936 to 1959, and was a special consultant there until 1969, when he retired.

SIXTY-YEAR PINS

Sixty-Year Pins will be presented to the following members who received Fifty-Year Pins in 1963:

Androscoggin County

RALPH A. GOODWIN, SR., M.D. – Dr. Goodwin was President of the Maine Medical Association in 1949-1950, President-elect in 1948-1949, Council Chairman in 1947-1948 and, prior to that, had served as Councilor for the Second District. He was graduated from Bates College and received his medical degree from Harvard Medical School in 1913, interned at the Rhode Island Hospital and took postgraduate courses at Massachusetts General Hospital and Central Maine General Hospital in Lewiston. Dr. Goodwin located in Auburn in 1916 and has been affiliated with the Central Maine General Hospital as adjunct surgeon from 1923 to 1932, visiting surgeon from 1932 to 1948 and consulting surgeon from 1948 to the present time. He also served as Medical Director for the Androscoggin County Civil Defense. Dr. Goodwin is a native of Danforth, Maine.

Dr. Goodwin

Dr. Melnick

Cumberland County

JACOB MELNICK, M.D. – Dr. Melnick, a native of Vilno, Lithuania, now resides in Portland. He attended schools in Lithuania, New Hampshire and Massachusetts and received his medical degree from the College of Physicians and Surgeons in Boston in 1913. Dr. Melnick interned at the Brockton Hospital in Massachusetts and then located in Portland. He is now retired.

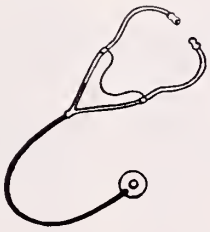
ALBERT W. MOULTON, SR., M.D. – Dr. Moulton, a native of South Portland, was graduated from Bowdoin College in 1909 and received his medical degree from Bowdoin Medical School in 1913. He interned at the Maine General Hospital and took a postgraduate course at the Manhattan Eye, Ear and Throat Hospital. Dr. Moulton has practiced in the Portland area since 1913 and has been affiliated with the Maine General Hospital, the Maine Eye and Ear Infirmary, the Maine Medical Center, the Mercy Hospital and the Webber Hospital.

Dr. Moulton

Dr. Reynolds

Kennebec County

RALPH L. REYNOLDS, M.D. – A native of China, Maine, Dr. Reynolds now resides in Waterville where he has practiced Obstetrics and Gynecology since 1914. He was graduated from Colby College in 1906, received his medical degree from Harvard Medical School in 1913, interned at the Boston City Hospital, served a residency at the Boston Lying-In Hospital and took postgraduate courses at the American College of Surgeons. His hospital appointments include the Seton Hospital and the Thayer Hospital in Waterville. Dr. Reynolds is a member of several professional organizations and has contributed many professional papers to *The Journal of the Maine Medical Association.* During World War I, he served with the Voluntary Medical Reserve Corps and received the Selective Service Medal.



Maine Heart Association Notes
20 Winter Street, Augusta, Maine

The Assessment of Myocardial Infarction With Serum Enzyme Determinations

WILLIAM E. SHELL, M.D.*

Elevated serum enzyme activity has become a cornerstone in the diagnosis of acute myocardial infarction. The absence of elevations when carefully sought for is an excellent criterion for exclusion of the diagnosis. Recently, investigative effort regarding serum enzyme elevations in patients with acute myocardial infarction has been directed toward the use of such determinations as indices of prognosis and specifically as a means by which the extent of ischemic myocardial injury can be quantified. Rapidly accumulating evidence suggests that prognosis after acute myocardial infarction is related to the extent of myocardial necrosis and that this in turn is reflected quantitatively by the magnitude of alterations of serum enzyme activities.

In experimental studies in the 1950's, it became clear that a general relationship existed between peak serum enzyme activity following coronary occlusion and the extent of myocardial necrosis assessed morphologically. Experimental myocardial infarction produced in open chest or closed chest preparations consistently led to elevated serum enzyme activity in serum. In general, small infarcts were associated with low peak levels of SGOT and LDH and large infarcts were associated in high peak levels. However, peak levels of these and other enzymes exhibited considerable variation in animals with infarcts of comparable size; hence, in individual animals determination of peak enzyme activity does not permit reliable estimation of infarct size measured pathologically. This variation is due in part to the effects of surgical interventions utilized in producing the experimental infarction on the activity of serum enzymes, biological variation in the rate of enzyme release from ischemic myocardium, and nonuniform ischemic injury within the infarct itself.

Despite these limitations, considerable evidence indicates that the magnitude of peak serum enzyme activity following myocardial infarction presages prognosis. After spontaneous myocardial infarction in man SGOT begins to rise, usually within 12 hours, and reaches a peak level within 24-36 hours. Peak SGOT values greater than 300 Karmen units has been associated with adverse prognosis and with a mortality of 40% within the first six weeks. On the basis of autopsy studies, it is apparent that a linear relationship exists between gross morphologic estimates of infarct size (small, medium, and large) and the magnitude of serum enzyme elevations. Thus, patients dying with small infarcts exhibited an average peak SGOT of 100 K.U. compared to 180 K.U. in those with medium infarcts and 450 K.U. in those with large infarcts. It is likely, that hepatic release of SGOT, reflecting congestive heart failure, contributes to the marked elevations seen in patients with large infarcts.

Serum lactic dehydrogenase (LDH) activity bears a similar relationship to infarct size. Peak LDH levels of greater than 150 U/ml are associated with increased risk and apparently large infarcts. A level of serum alphahydroxybutyric acid dehydrogenase (HBD), reflecting the activity of isoenzymes of LDH typical of those seen in heart muscle, exceeding 800 IU/ml has been associated with mortality rates of 30% while a level less than 800 IU/ml has been associated with a mortality rate of 15% or less. It is important to recognize that LDH activity in serum frequently reflects extraneous sources of enzyme, particularly the red cells which release large quantities of LDH into serum with even minimal hemolysis.

Serum creatine phosphokinase (CPK) activity appears to be a good index of prognosis. Peak serum CPK activities exceeding 600 mlU/ml are associated with mortality rates greater than 30% while values less than 600 mlU/ml (12 times normal) are associated with a risk of only 12%. It has been found that peak values less than 300 mlU/ml are associated with mortality rates of only 2%. Results of CPK determinations are dependent upon the specific assay employed and become meaningful only when the assay is performed with careful attention to technique.

Morbidity as well as mortality appeared to be related to the magnitude of peak serum enzyme activities following myocardial infarction. Major arrhythmias such as ventricular tachycardia, ventricular fibrillation, and A-V block occur much more commonly in patients with high peak serum enzyme activities. Severe hemodynamic abnormalities also appear to be more common in patients with high enzyme values. Moreover, the hemodynamic abnormalities may persist for sev-

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Prepared by the Maine Heart Association for this Journal.

eral months or indefinitely in those patients with high serum enzyme values associated with an episode of acute infarction. Patients with marked serum enzyme elevations are likely to show increases in left ventricular end diastolic pressure during the acute phase and frequently progression of these abnormalities during succeeding months. Wall motion disorders are also more common in those patients with marked elevation of serum enzyme activity. It appears likely that the electrophysiological, physiological, and prognostic features associated with marked elevation of serum enzyme activity after acute myocardial infarction are all related to infarct size, in turn reflected by the magnitude of peak enzyme elevation.

The peak level of enzyme activity observed after acute myocardial infarction depends on several factors including: 1) frequency of sampling, 2) the rate of enzyme release from myocardium, 3) the rate of enzyme clearance from the circulation, 4) the volume into which the enzyme is distributed, 5) contributions from extraneous non-cardiac sources of enzyme, and 6) infarct size. In order to relate enzyme changes appropriately to infarct size and to prognosis, these factors must be taken into consideration. In patients with acute myocardial infarction, samples for enzyme determinations at least every 12 hours are helpful in assessing trends and in determining the actual peak level. Intramuscular injections should be kept to a minimum in order to reduce the likelihood that extraneous sources of enzyme will cloud the picture. Blood samples must be obtained with thorough precautions to avoid hemolysis. Isoenzyme determinations should be employed to facilitate interpretation of elevated enzyme activities.

Since the observed levels of enzyme activity in blood in part reflect infarct size, serum enzyme analysis may offer a more precise means of estimating the extent of myocardial injury associated with a specific episode of infarction. We have recently utilized serial changes in serum CPK activity to measure infarct size in patients. The approach is based on a mathematical model which takes into account the rate of CPK release, CPK clearance, and the plasma volume into which CPK is distributed. In animal experiments, we have observed a close correlation between infarct size assessed from serum CPK changes analyzed with the use of this model and infarct size measured directly by analysis of myocardial CPK depletion. In patients, assessment of infarct size by serial serum CPK analysis has served as a reliable index of subsequent mortality and morbidity. In patients with estimated infarct size exceeding 50 CPK-gram-equivalents, acute mortality (within 30 days) was 36% compared to 3% in those patients with infarct size less than 50 CPK-g-eq. Long term mortality and morbidity was similarly related to CPK-infarct size. In patients followed for six to twelve months, 67% of those with infarct size exceeding 65 CPK-g-eq were either dead or Class III or IV. Thus, analysis of serial enzyme changes has proven useful in the assessment of infarct size, in turn a valuable index permitting separation of patients according to risk.

Summary

Peak SGOT, LDH, and CPK levels are related to prognosis after acute myocardial infarction. Peak serum enzyme activity and prognosis correlate probably because they are both related to infarct size. Infarct size can be estimated reliably in patients by mathematical analysis of serial serum CPK changes. This approach permits improved assessment of risk in specific patients with acute myocardial infarction and should prove valuable not only in facilitating the management of individual patients but also in conducting clinical trials to assess therapeutic mortalities with potential risk. Clinical enzymology is no longer merely a tool for the recognition of acute myocardial infarction but is rapidly becoming applicable as an important means for assessing the severity of the infarction and estimating prognosis reliably.

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An Incident of Zectran* Poisoning

ERNEST M. RICHARDSON and ROBERT I. BATTEESE, JR. **

Use of organophosphate and carbamate insecticides has greatly increased in the last five years due to governmental restrictions or outright banning of the more persistent chlorinated hydrocarbon chemicals such as DDT, Dieldrin, Endrin, Heptachlor and Toxaphene. Although the newer materials have the advantage of rapid environmental breakdown, they are considerably more toxic for humans exposed to the chemical in liquid or vapor form. Exposure may occur by direct absorption through unprotected skin as well as by respiratory and oral routes. Persons neglecting to wear protective clothing and a respirator are therefore prone to poisoning. From a medical point of view, cases of human intoxication by these insecticides often pose difficult problems because of the rapid onset and progression of symptoms, similarity of symptoms to other diseases, and unfamiliarity with specific antidotal treatment.

Zectran is a cholinesterase inhibiting, carbamate insecticide used to control certain insects attacking turf, ornamentals and forests. Although it is highly toxic when exposure occurs through oral or respiratory routes, the hazard from dermal exposure is slight and the chemical is considered quite safe when applied at recommended rates.

Only one case of Zectran poisoning can be found in the literature and it reported the death of a young nursery worker who apparently committed suicide by ingesting a quantity of the pesticide concentrate.[1] The incident described herein is believed to be the first report of a person being occupationally poisoned while applying the chemical according to label directions. It occurred in June 1972 during the aerial spraying operation by the Maine Department of Forestry for Spruce Budworm control. Over 512,000 acres of northern Maine woodlands were sprayed with Zectran at the recommended dosage rate of 0.15 pounds active ingredient per acre.

A co-pilot in one spray aircraft became ill during his second flight of the morning. The cause was attributed to a pin-hole leak in a high pressure pump line which was located twelve feet behind the co-pilot's seat and which emitted a fine aerosol spray into the fuselage. Duration of exposure was estimated at 110 minutes, the flying time of that morning. Midway through his second flight he began to experience many of the classical symptoms of organophosphate and carbamate insecticides.[2,3] General weakness, severe headache, stomach cramps, and a metallic taste in the mouth were initially noted and protective respirators were donned at this time. Symptoms progressed and within minutes he experienced a feeling of extreme drowsiness, temporary blurring of vision, loss of depth perception and difficulty in breathing.

After landing the aircraft, the pilot and others removed the co-pilot from the plane. He had no sense of balance and could not stand or walk. Headache and stomach cramps increased in severity and he shook uncontrollably. While in transport to the hospital, his hands and arms became numb and paralyzed and his speech became slurred. He was however, mentally alert of the happenings around him.

A hospital staff, previously briefed on the medical aspects of this insecticides, observed miosis, flushed face and muscle twitching. Atropine Sulfate, 1/75 gr. IV, was administered immediately with a second injection subcutaneously forty minutes later. Most symptoms subsided quickly and an electrocardiogram revealed normal readings. Three hours after admittance, the patient was released from the hospital and returned to his quarters where he was kept under close observation by the project health officer and fellow crew members. A headache and general feeling of weakness persisted and he remained in bed for the remainder of that day.

*Zectran is the registered trade name of Dow Chemical Co. for 4-Dimethylamino-3, 5xylyl N-methylcarbamate.

**Pesticide Residue Analyst and Pesticide Project Coordinator, respectively, Maine Pesticide Program, Public Health Laboratory. Served as health officers for Spruce Budworm Project conducted by Maine Department of Forestry. The Maine Pesticides Program is supported by Contract No. 68-02-0547 from the Environmental Agency.

Cholinesterase activity in a blood sample drawn ten minutes after the first atropine injection was severely depressed. Since the inhibition reaction by carbamates is readily reversible upon cessation of exposure, the activity had returned to normal after three days. The co-pilot was then allowed to resume his duties and he did not experience any further difficulty during the remaining spray project.

The above incident illustrates that pesticide poisoning can and does occur in Maine and the frequency of incidents will likely increase as the shift to greater use of organophosphates and carbamates continues. The co-pilot was fortunate in receiving prompt treatment since the hospital staff were trained in handling pesticide poisoning cases and the spray crew who took him to the emergency room reported that he had been exposed to Zectran. Oftentimes, physicians experience difficulty in making the proper diagnosis because they are unfamiliar with the symptoms and no direct evidence exists linking the illness to pesticide poisoning. For these reasons and because many of the symptoms mimic other diseases, it appears desirable to present the following information so medical personnel may familiarize themselves with the symptoms and treatment procedures.

According to Davis et al,[2] "The clinical manifestations of organophosphate and carbamate pesticide poisoning are well described and include miosis, weakness and collapse, muscular fasiculations, goose flesh, diaphoresis, pulmonary edema, nausea, vomiting and diarrhea. Unfortunately, not every patient evidences a set pattern of clinical response when first seen by a physician. The clinical signs may be confused with encephalitis, brain injury, hypertensive encephalopathy, pneumonitis, gastroenteritis, asthma and congestive heart failure, to name a few." When persons fail to respond to normal treatment measures for these diseases, one should then consider the possibility of pesticide involvement.

The correct order of treatment is important and has been outlined by Hayes[3] as follows:

I. Severe cases —
   (1) Artificial respiration, if required, preferably by mechanical means.
   (2) Atropine Sulfate, 2 to 4 mg. (1/30 to 1/15 grain) intravenously as soon as cyanosis is overcome. Repeat at 5 to 10 minute intervals until signs of atropinization appear, (dry, flushed skin and tachycardia as high as 140 per minute).
   (3) Decontamination of the skin, stomach and eyes as indicated.
   (4) Symptomatic treatment.

II. In the more usual cases —
   (1) Atropine Sulfate, 1 to 2 mg. (1/60 to 1/30 grain), if symptoms appear. If excessive secretions occur, keep the patient fully atropinized. Give atropine sulfate every hour up to 25 to 50 mg. in a day.
   (2) Decontamination of the skin, stomach and eyes as indicated.
   (3) Symptomatic treatment.

Important points:
   (1) Atropine is used in much larger doses than normal.
   (2) If exposure was primarily by a dermal route, it is extremely important to wash the skin thoroughly.
   (3) If you are certain an organophosphate was involved, 2 PAM[3] may also be used. Do not use if there is any chance carbamates were involved.
   (4) Keep close watch of patient for at least 24 hours to be sure patient does not suffer relapse.
   (5) Draw a 7-10 cc heparinized blood sample for a cholinesterase determination prior to administering atropine.

In the event of a pesticide poisoning incident, the Togus Poison Control Center should be contacted to obtain the latest information on proper treatment; they may be reached at 623-8411. Also contact the Public Health Laboratory, 289-2727 for information and assistance in a field investigation of the incident. The laboratory is also able to provide back-up cholinesterase monitoring to confirm organophosphate carbamate poisoning and is fully equipped for determining pesticides in tissue samples and in fluids from gastric lavage, urine and blood.

Introduction of the organophosphate and carbamate pesticides into the southern states resulted in many tragic incidents until the physicians became experienced in diagnosing and treating these poisoning cases. It is hoped that this case report and accompanying information will enable all medical and related personnel encounterine pesticides poisonings to achieve satisfactory patient recovery.

REFERENCES

1. Reich, G. A. and J. O. Welke. 1966. Death due to a pesticide. New. Eng. J. Med. 274: 1432.
2. Davis, J. H., J. E. Davies and A. J. Fisk. 1969. Occurrence, diagnosis, and treatment of organophosphate pesticide poisoning in man. Ann. N.Y. Acad. Sci. 160: 383-392.
3. Hayes, W. J. 1963. Clinical Handbook on Economic Poisons: Emergency Information for Treating Poisoning. Publ. Hlth. Serv. Rep. No. 476.

Necrologies

Rodolphe J. F. Pomerleau, M.D.

1907-1972

Word has been received that Dr. Rodolphe J. F. Pomerleau died on October 18, 1972 following a long illness.

Dr. Pomerleau was born in Waterville, Maine on August 22, 1907, son of Vilbon and Wilburge Pomerleau. He was graduated from Waterville High School in 1925, Colby College in 1929, received his medical degree from Boston University Medical School in 1934 and took a postgraduate course at St. Joseph's Hospital in Lowell, Massachusetts. Dr. Pomerleau had practiced in Waterville since 1935. During World War II, he served in the Army Medical Corps.

He was a member of the Kennebec County Medical Association and the Maine Medical Association.

Surviving are a sister, Gabrielle Guite of Waterville and three nephews.

Ward A. Albro, M.D.

1919-1973

Dr. Ward A. Albro, 53, of Houlton, Maine died on May 5 of a sudden coronary while horseback riding.

He was born in South Royalton, Vermont on July 4, 1919, son of Bertram A. and Sfa W. Albro.

Dr. Albro was graduated from Tufts College in 1941 and received his medical degree from Tufts University School of Medicine in 1944. He interned and was an assistant resident surgeon at the Massachusetts Memorial Hospital, did postgraduate work in pathology at Dartmouth Medical School and postgraduate work in anesthesia at the Maine Medical Center. He served in the U. S. Air Force for three years and was chief of surgery at Dow Air Force Base in Bangor.

He was a member of the Aroostook County Medical Society, the Maine Medical Association, the American Society of Anesthesiologists, the American Academy of General Practice, the Maine Society of Anesthesiologists and the Maine Society of Clinical Hypnosis. Dr. Albro had served on the surgical staff at the Eastern Maine Medical Center and the Waldo County General Hospital, and the anesthesia staff at St. Joseph's Hospital and the Augusta General Hospital. At the time of his death, he was chief of staff of the Houlton Regional Hospital. He was also a member of the Maine Horse Association and was president of the Aroostook Riding Club.

Surviving are his widow, Judith Albro; four sons, Carl of Columbus, Ohio, David and Eric, both of North Northampton, Massachusetts, Todd of Houlton; two daughters, Mrs. Edward Harvey and Miss Jan Albro, both of Northampton, Massachusetts; one sister, Mrs. Arthur Cutter of Rochester, New Hampshire and two grandchildren.

Joseph R. Ridlon, M.D.

1882-1973

Dr. Joseph R. Ridlon, 91, of Gorham, Maine died on June 2 after a brief illness.

He was born in Gorham on April 18, 1882, son of Francis A. and Evelyn M. Ridlon.

Dr. Ridlon was graduated from Gorham High School in 1899, Bowdoin College in 1903 and received his medical degree from Bowdoin Medical School in 1906. He interned at the Maine General Hospital and took postgraduate courses at London School of Tropical Medicine. He had hospital appointments at the Marine Hospital in Savannah, Georgia, New York, New Orleans, Galveston, Texas and Detroit, Michigan. Dr. Ridlon served in the U.S. Public Health Service and U.S. Navy and retired with the rank of Colonel.

He was an honorary member of the Cumberland County Medical Society and the Maine Medical Association, receiving a 55-year pin in 1964 and a 60-year pin in 1969. He was also a member of the American Medical Association, the American Board of Preventive Medicine and Public Health and a fellow of the American College of Physicians. Dr. Ridlon was active in local civil defense and Red Cross activities from 1946 to 1956 as an instructor in first aid and medicine.

He was a member of the Maine Hospital Advisory Council from 1953 to 1962.

Surviving are a daughter, Mrs. Evelyn R. Adams of Gorham; three grandchildren and five great-grandchildren.

County Society Notes

CUMBERLAND

The 377th meeting of the Cumberland County Medical Society was held on April 24, 1973 at the new Holiday Inn on Spring Street in Portland.

One hundred three members and guests were present. Our guests included Drs. George W. Wood, III, Linus J. Stitham, John B. Madigan, H. Carl Amrein, Richard C. Leck, Donald L. Anderson, George W. Bostwick and Richard T. Chamberlin, all of whom are members of the State Executive Committee, and Drs. Gilbert R. Grimes and Herbert J. Wright, Jr. from the Androscoggin County Medical Association.

Following a well-attended social hour, an excellent roast beef dinner was served by the management of Holiday Inns, thus redeeming themselves for last month's poor performance.

Following the dinner, the business meeting was called to order. Reading of the minutes of the last meeting was omitted. A second reading of the application of Dr. Fred Ginn was made and Dr. Ginn elected to membership.

First reading of the application of Dr. Donald Bitterman for junior membership was made.

Resolution on the death of Dr. Edward Greco, Sr. was presented by Dr. Eugene E. O'Donnell. The resolution was unanimously passed and will be attached to our records.

Dr. Douglas R. Hill announced that the chiropractic hearing will be held in Augusta tomorrow at 1:30 p.m. and urged attendance by as many members as possible. Our second television show will be presented sometime during the month of May. The exact date and time have not yet been determined.

Our next meeting will be the annual meeting. It will be held at Valle's on May 17. The election of officers and reports of all committees will be presented.

The President of the Maine Medical Association, Dr. George W. Wood, III, then introduced our guest speaker, Dr. Raymond T. Holden. Dr. Holden is a second term member of the American Medical Association Board of Trustees.

Dr. Holden discussed the AMA position on several major items. The first topic was Medi-Credit which is the bill introduced and sponsored by the AMA and would allow multiple options as to methods of health care. It would not drastically restructure the whole system of medical care. He compared this to the Kennedy-Griffith Bill which has an estimated annual cost of $80 billion.

The topic of HMO was next discussed. Basically, these are groups financing medical care by pre-payment. This is nothing new. There are no real experts in this field and Senator Kennedy has introduced a bill which would blanket the country with HMO to the cost in excess of $1 billion. The Administration's plan has been greatly modified.

Concerning the union movement, it is Dr. Holden's feeling that there is nothing which the unions can accomplish which cannot be accomplished through the existing county, state and national medical societies. At this time, there are three cases being presented before the courts relative to the rights of physicians to practice and determine fees, all of which have been actively supported by the Association. The unions have no special legal tools which the AMA does not have.

Following Dr. Holden's presentation, there were numerous questions from the floor.

Following the discussion, the meeting was adjourned at 9:30 p.m.

The annual meeting of the Cumberland County Medical Society was well attended. Following an excellent dinner which was very efficiently served, a lengthy business meeting ensued.

The CCMS informational answering service is now functioning. The phone number will be listed under CCMS in the new telephone directory in June. The second television show will be aired June 8 at 8:00 p.m. over Channel 13. The stars of this month's presentation will be Drs. Carroll Hiebert, Hill and Thorpe. Don't miss it!

The annual reports of standing committees were presented. One concludes from these reports that there is a great deal of on-going activity concerning many medical topics of great importance to each of you. This Society owes a large measure of gratitude to the Chairmen of the various committees who devote a great deal of time to their projects. Dr. Hill presented the President's report and summarized the past year's activities. We are also indebted to Doug for the fine service that he has provided.

The following list consists of the delegates and alternates.

One Year Delegates: Drs. John R. Davy, Irving J. Poliner, Peter B. Webber, Robert G. Sommer, Stephen E. Monaghan and Douglas R. Hill.

One Year Alternates: Drs. Harry A. Bliss, William J. Hall, III, Wesley J. English, Hugh P. Robinson, Paul Maier and Kirk K. Barnes.

Two Year Delegates: Drs. Ronald J. Carroll, George I. Geer, Jr., John F. Gibbons, Walter B. Goldfarb, William L. MacVane, Jr. and Donald F. Marshall.

Two Year Alternates: Drs. Newell A. Augur, Jr., Elio Baldini, Carl A. Brinkman, John T. Dinan, Jr., Robert H. Pawle and Roland G. Ware, Jr.

The one year appointments will complete their terms in June of 1974. The two year appointments commence in July of 1973 and complete their terms in June of 1975.

Dr. Lawrence Crane, Chairman of the Nominating Committee, presented the nominations for the elective offices. Dr. Douglas R. Hill was re-elected to a second year as President of the Society. Dr. Winton Briggs was re-elected as Vice-President. Dr. Morrill Shapiro was elected to a five-year term as a member of our Professional Ethics Committee and Dr. Harry A. Bliss was elected to a three-year term on our Executive Committee.

There will be no regularly scheduled meetings of the Society until September. The Executive Committee does meet regularly during the summer. We hope to make our first meeting in September a special event. Any suggestions from the membership will be greatly appreciated.

A reminder! The meeting of the Maine Medical Association will be held in Kennebunkport at the Shawmut Inn, June 16-19. For those of you who have not attended previously, we welcome your support at the House of Delegates meeting and urge your attendance at the scientific sessions as well.

ALFRED E. SWETT, M.D., *Secretary*

PENOBSCOT

The March 1973 meeting of the Penobscot County Medical Society was held on March 20 at the Tarratine Club, Bangor, Maine with 39 members in attendance.

The meeting was opened by the President, Dr. Benjamin L. Shapero, who then introduced the members of the panel comprised of Drs. John A. Ordway, Linus J. Stitham, Mr. William Shook, and Rev. John Miller, who then embarked upon the discussion of the night's topic "Health Care Delivery Systems." Each panelist spoke in turn and provided his remarks on this topic of discussion. Mr. Shook outlined the history and evolution of Health Care organizations within the community, their design, and origins, and the development of them by predominantly non-physician lay personnel with the approval and support of the Federal and State Governments. Rev. John Miller, stated that the evolution of Health Care Delivery Systems has been in the direction of a bigger and larger administration of the organiza-

tion involved, but at the same time becoming less and less in touch with the people with whom they are intended to serve. Dr. Stitham made various observations with regards to the day-to-day operation of these organizations at the small community level. Finally, Dr. Ordway, moderating the panel throughout the discussion, provided his own observations of the existing and possible future Health Care Delivery Systems. A discussion followed the above presentations which was both broad and diverse. These discussions were primarily concerned with the many and rather detailed organizations presently existing which are each involved in the delivery of medical care without advice and direction of the medical community as a whole.

The business meeting of the Society then followed the panel discussion described above.

The minutes of the February 1973 meeting were read and approved.

There were no committee reports.

There was no old business.

A discussion regarding a tentative proposal by one or more County Societies to come before the April meeting of the House of Delegates to raise the yearly dues of the Maine Medical Association for the purpose of employing one or more persons to become involved and participate in keeping the Maine Medical Association and its County Societies abreast and informed of the direction of Health Care Organizations and Delivery Systems on a State and local basis ensued. The delegates were instructed to support such a proposal if one was forthcoming at the April meeting of the House of Delegates.

A request was made for the Society to take a position in regard to a recent bill introduced by Senator Kenneth MacLeod regarding the practice of chiropractic in the State of Maine. There was no final action taken on this request.

Application for membership in the Penobscot County Medical Society of Dr. Eugene R. Lucier was read, voted upon by the members present and approved by a unanimous vote.

There being no more business, the meeting was then adjourned.

PHILIP G. HUNTER, M.D., *Secretary*

ANDROSCOGGIN

The meeting of the Androscoggin County Medical Association was held at Steckino's Restaurant, Thursday, April 19, 1973. The meeting was called to order at 8:00 p.m. by the President, Dr. Gilbert R. Grimes, with 30 members present.

The minutes of the March meeting were accepted as read. Several letters to the Association were accepted and acted upon: letter from Dr. Henry C. Thacher requesting continuance of his membership during one year absence to attend postgraduate education was approved; letter from Dr. Merton N. Flanders requesting continuance of his membership even though he is no longer in active clinical practice was discussed and will be researched and forwarded to Maine Medical Association requesting transfer to affiliate membership; letter from M.M.A. to Dr. Philip L. Archambault indicating his transfer to affiliate membership.

Application of Dr. Kenneth Wolf, sponsored by Dr. Jou S. Tchao, was accepted and forwarded to the credential committee for later action.

Under old business, Dr. Charles A. Hannigan accepted reappointment as State representative as Advisor to Maine Association of Medical Assistants. Dr. Grimes will appoint a member as District representative to Maine Association of Medical Assistants.

Dr. Paul M. Beegel was appointed by President Grimes to the representation of A.C.M.A. to the Tri-county Comprehensive Health Planning Agency. There was considerable discussion of Dr. Hannigan's proposal to seek legal counsel to represent this body and its respective members. The proposal was accepted in principle and will be fully researched by Dr. Grimes and the Executive Committee with later report to the A.C.M.A. of feasibility.

During discussion of new business, Dr. Donald L. Anderson presented a full discussion of the February Executive Committee meeting of the M.M.A.

Dr. Behzad Fakhery presented copies of the business conducted at the interim meeting of the House of Delegates of the M.M.A. The delegates of this County were instructed by vote of the Association to support the Penobscot County resolution dealing with chiropractic in the State (Maine), and the Resolution proposed by the Committee on Continuing Education.

The meeting was then turned over to Dr. Gerard L. Morin, program director, who introduced Mr. Stephen Marsden, executive director of Tri-County Health Planning Agency. Mr. Marsden explained the position of the Tri-County Health Planning Agency in regards to its proposals for changing Health Care Delivery in the Lewiston-Auburn area. An enlightening discussion ensued.

The meeting was adjourned at 9:50 p.m.

RICHARD M. SWENGEL, M.D., *Secretary*

KENNEBEC

The Kennebec County Medical Association held its annual husbands' and wives' dinner meeting at the Augusta Country Club on May 17, 1973. Following an enjoyable buffet, the business meeting was opened by the President, Dr. Richard R. Dole. A motion to dispense with the reading of the minutes was duly made and seconded. Dr. Dole then introduced Dr. John D. Denison who outlined the family practice residency program to be conducted by the Family Medicine Institute at the Augusta General Hospital.

Dr. Dole then introduced the main speaker of the evening, Dr. George W. Wood, III, who as president of the Maine Medical Association, gave an informative presentation of recent and current Association activities. He outlined some of the efforts to establish a PSRO for Maine; gave an "up-to-date" report of some medical legislative activities, including the physician's assistant and chiropractic questions; and discussed recent developments in establishing a medical school in Maine. Dr. Wood's interesting comments were well received by the good-sized gathering.

The meeting was adjourned at 9:30 p.m. or thereabouts.

KEVIN HILL, M.D., *Secretary*

Letter to the Editor

To the Editor:

I am editing a book on renown and notable physicians and their faith.

I am interested in obtaining contributors who have a special knowledge of the faith and/or religion of one or more notable and outstanding physicians. I am considering such physicians as Sir William Osler and Sir William Fleming; however, the notable physicians could still be alive.

Anyone interested in this project or who would suggest renown physicians to write about may contact me at the following address:

CLAUDE A. FRAZIER, M.D.
4-C Doctor's Park
Asheville, N.C. 28801

The Journal
of the
Maine Medical Association

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100 Years of Internships

This issue of the Maine Medical Journal, the first to be designated as the Maine Medical Center House Staff Number, commemorates the 100th anniversary of the Maine Medical Center House Officer Program.

In 1874, Erastus Holt and George W. Libby, both graduates of the Bowdoin Medical College, joined the hospital as the first "house pupils" as interns were then called. The line of continuity in house staff education has been a continuous one for the past 100 years, albeit a bit thin at times.

When the medical school in Portland closed its doors in 1921 under the impact of the Flexner Report, the hospital continued its intern program accepting graduates of other medical schools, particularly from Tufts. The teaching program since then has increased both in numbers of house staff as well as variety of programs at an intern and resident level. Increasing numbers of medical students in their third and fourth year have rotated through the various teaching departments and divisions.

Nineteen hundred and seventy-three has been a peak year with a total of 110 individuals spending all or part of the year at the Maine Medical Center. These consisted of 47 residents, 15 interns, 32 fourth year students and 16 third year students, the latter spending their entire third year at the hospital. The benefit to the State is clearly demonstrated by the fact that over 50% of our alumni go into practice in the State of Maine.

ALBERT ARANSON, M.D.
Chief, Department of Internal Medicine
Director of Medical Educaion
Maine Medical Center
Portland, Maine 04102

Traumatic Dissecting Aneurysm of the Abdominal Aorta

ROBERT BLUTE, B.S.* and FERRIS RAY, M.D.**

In recent years, increasing numbers of reports have described injuries to the *thoracic* aorta associated with blunt trauma. Half of these have been associated with highway accidents attributed to deceleration and shearing of the aorta at its points of fixation within the thorax. In 1965, Gnu and Konstam[1] reported successful treatment of a patient with dissecting aneurysm of the *abdominal* aorta following blunt trauma and only eight additional cases with this unusual injury have since been reported by other authors. Although automobile accidents were responsible for these injuries in eight of the nine reported cases, this was the only feature in common with thoracic aortic injuries in terms of the pathological sequence of events, diagnosis, treatment, and prognosis. Because early recognition of the injury determines prognosis and results of treatment following such injury, it seems worthwhile to report a tenth case in whom delayed diagnosis and treatment contributed to death.

CASE REPORT

E. D., a 78-year-old female, was wearing a lap seat belt when driving her automobile and involved in a moving accident with another vehicle. The factors of involved force could not be obtained but are suggested by the ruptured spleen and traumatic rupture of the diaphragm sustained by the driver of the other vehicle. E. D. was taken to a nearby hospital where facial lacerations were sutured and, because multiple facial fractures were identified, she was transferred to a second hospital. On arrival, her blood pressure was 130/70 mm Hg in the arm, pulse rate was 110 per minute, and she complained of pain in the lower back and extremities. Examination revealed motor paralysis of both lower extremities, decreasing sensation from groin to toes, left fibular fracture, and multiple facial fractures. A diagnosis of spinal cord injury was made and the patient transferred to the Maine Medical Center nine hours after injury. An astute Surgical Resident confirmed the above findings but also noted reflexes of the lower extremities and absent femoral pulses. The blood pressure was unchanged and the abdomen was soft with bowel sounds present. No bruit could be heard over the abdominal aorta. The lower extremities were pale and cool with decreased pinprick sensation from toes to the umbilicus. Laboratory studies indicated a hemoglobin concentration of 10.6 gm%, hematocrit 31.4%, and normal serum electrolytes. X-ray studies of the chest, spine, and abdomen were within normal limits but the skull films showed multiple malar fractures. Intravenous pyelogram indicated normal renal function bilaterally and cystogram showed no evidence of urinary bladder injury. A right transaxillary aortagram demonstrated complete occlusion of the abdominal aorta distal to the renal arteries which were patent. A diagnosis of traumatic occlusion of the abdominal aorta was made and an operation performed 14 hours after the accident.

Initial inspection of laparotomy revealed hematoma in the small bowel mesentery and the retroperitoneal areas in the mid-abdomen. The aorta and iliac arteries appeared normal in caliber but with blue discoloration and absent pulsations below the level of the inferior mesenteric artery. Aortotomy at this level revealed an intimal tear posteriorly with distal dissection into the common iliac arteries.

The dissected intima was displaced into the lumen causing complete aortic occlusion. Marked atherotic change was present in the involved segment of the aorta. An aorto-femoral bypass dacron graft was inserted with restoration of pedal pulses. However, immediately after removal of the occlusion vascular clamps to restore flow, the blood pressure became unstable in association with cardiac arrhythmia and a serum potassium concentration of 9.0 meq/L. She expired two hours postoperatively with hypotension, arrhythmia and hyperkalemia not responding to treatment. Autopsy examination confirmed the presence of marked arteriosclerotic changes in the abdominal aorta but demonstrated no acute myocardial changes.

DISCUSSION

The mechanism of injury, the type of lesion sustained, and the clinical courses of the ten reported cases were remarkably similar and are summarized in Table 1. All ten patients sustained blunt trauma to the abdomen during acute deceleration with partial or complete disruption of the abdominal aortic intima followed by mural dissection and occlusion of the aortic lumen, its major side branches, or both. In contrast to the shearing force causing a similar injury to the thoracic aorta subjected to blunt trauma, the abdominal aortic lesion results from compression of the aorta between the wounding object such as a steering wheel, seat belt, or other solid object and non-yielding vertebral column. The severity of aortic atheromatous change and the magnitude of force apparently determine whether complete disruption of the aorta occurs or intimal tearing only with subsequent mural dissection.

The prognosis of traumatic dissecting aneurysm of the abdominal aorta compares favorably with that of ruptured thoracic aorta (> 70% mortality) or ruptured abdominal aorta (70% mortality).[9] Seven of the ten reported cases survived, including the patient of Rybak and Thomford who did not have primary vascular repair. One death followed proximal dissection of the renal and mesenteric arteries without aortic occlusion. Laparotomy was performed with extensive resection of gangrenous bowel, but the vascular lesion was not recognized

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TABLE 1

SUMMARY DATA OF TEN REPORTED PATIENTS WITH TRAUMATIC ABDOMINAL AORTIC DISSECTING ANEURYSMS

Author	*Age Sex*	*Vehicle*	*Seat Belt*	*Femoral Pulses*	*Leg Pinprick Sensation*	*Leg Motor Weakness*	*Operative Delay*	*Type Operation*	*Result*
Ngu Konstam 1965[1]	37 / F	Surf Board	No	Absent	Reduced	Present	30 hours	Thromboend-arterectomy	Foot drop; amputation of three toes.
Tomatis & Perry 1968[2]	27 / F	Auto	No	Absent	Reduced	Present	6 hours	Aorto-iliac graft	Full recovery
Rybak & Thomford 1969[3]	46 / F	Auto	Yes	Absent	Reduced	Present	9 months	None, early	Aorto-iliac graft; below knee amputation.
Hewett & Grablowsky 1970[4]	65 / M	Auto	No	Absent	Reduced	Present	12 hours	Aorto-femoral bypass graft	Died
David & Blumenberg 1970[5]	54 / F	Auto	No	Reduced	Reduced	Present	—	None	Died
Borja & Lansing 1970[6]	45 / M	Auto	No	Absent	Reduced	Present	< 6 hours	Thromboend-arterectomy	Full recovery
Thal & Perry 1971[7]	38 / M	Auto	No	Absent, R Present, L	Reduced Present	Present	< 6 hours	Thromboend-arterectomy	Full recovery
Thal & Perry 1971[7]	38 / F	Auto	Yes	Reduced	Present	Absent	< 6 hours	Aorto-iliac graft	Full recovery
Angel & Crosthwait 1972[8]	60 / M	Auto	Yes	Absent	Reduced	Present	< 6 hours	Aorto-iliac graft	Full recovery
Present Case	78 / F	Auto	Yes	Absent	Reduced	Present	14 hours	Aorto-femoral bypass graft	Died

until postmortem examination.[5] The second death occurred under circumstances very similar to those of the present case; release of the aortic clamps and restoration of flow was abruptly followed by hypotension, bradycardia, and death.[4] In both instances, the stability of blood pressure and cardiac rhythm prior to release of aortic clamps and adequate volumes of intravenous fluids before and during operation seemed to rule out hypotension due to hypovolemia caused by pooling in the re-opened vascular space of the lower extremities. The clinical impression in each case, however, was that release of depressor substance had occurred, as suggested by Malette, Armstrong, and Criscuolo.[10] Hyperkalemia with arrythmia was documented in the present case. It is interesting to note that in all cases where surgery was performed within six (6) hours of injury, full recovery occurred. There is an implication that the accumulation of metabolic and other noxious substances in extremities ischemic more than six hours may be lethal unless the initial venous drainage following restoration of flow is vented and discarded. Perhaps the rapidity of neurological and ischemic clinical changes is an indication of the presence or absence of adequate collateral circulation to prevent the accumulation of toxic metabolites. For example, the patient of Gnu and Konstam did not have restoration of flow until 30 hours after injury yet did not react unfavorably to release of aortic clamps.

Of particular interest is the nature of sensory and motor changes in all but one of the ten cases. The degree and level of changes were higher than could be attributed to peripheral nerve and muscle ischemia, suggesting that obstruction or spasm of lumbar arteries supplying the spinal cord may occur at least transiently. For example, the dissection in the patient of David and Blumenberg did not occlude the aortic lumen but undoubtedly involved lumbar arteries at that level. The patient of Angel and Crosthwait walked away from the accident but developed paralysis and anesthesia of the legs by the time he reached the hospital, changes more consistent with progressive dissection and cord ischemia than peripheral ischemia. Finally, the patient of David and Blumenberg had paraplegia immediately after the accident but developed partial return of function in the hospital preoperatively. Reversibility of neurological changes may be expected following early restoration of aortic flow in the absence of associated traumatic cord injury, as indicated by the patients in this series and by the report of Potter and Hopkins.[11]

Associated injuries were present in seven of the ten patients but were important prognostically only in the patient reported by Rybak and Thomford in intra-abdominal abscesses were found at

laparotomy three days after injury, necessitating deferred vascular repair. In all of the patients who presented initially with sensory and motor deficits in the lower extremities, spinal cord injury was suspected until absent or diminished peripheral pulses led to aortography and demonstration of an aortic lesion. The laparotomy finding of periaortic ecchymosis or hematoma between the renal and iliac arteries and absent distal aortic pulsation led to aortotomy, recognition and correction of the sub-intimal dissection by endarterectomy or graft.

Because of the importance of *early* intervention in obtaining reversible neurological changes and viable extremities, the features of early recognition are worth reiterating. The victim of blunt trauma to the abdomen should be examined for femoral diminution or absence of pedal pulses, especially if numbness, diminished pinprick sensation or motor weakness appears following injury. If pulse abnormalities are present, aortography should be performed promptly to exclude aortic disruption or dissection. Evidence of an aortic lesion in the presence of diminished or absent pulses and neurological deficit involving the lower extremities is sufficient indication for immediate operation, provided other life-threatening associated injuries have been resolved.

Summary

Ten patients with traumatic abdominal aortic dissecting aneurysms following blunt abdominal injury have been reported. The mortality of 30% was associated with failure of recognition of the aortic lesion in one case and with hypotension and cardiac arrhythmia in two cases following release of aortic clamps after correction of the aortic dissection. Both of the latter cases were operated 12 and 14 hours after injury, respectively. Consideration of venting the first venous effluent following restoration of flow is raised, particularly when restoration of flow occurs more than six hours after aortic occlusion. Morbidity in two surviving cases was associated with delay in operation and consisted of permanent foot drop in one case and ultimate below-knee amputation in the second case. The association of absent or diminished femoral and peripheral pulses with fixed or changing sensory and motor deficits of the lower extremities following blunt trauma to the abdomen is an indication for immediate aortography and correction of the probable aortic dissection of disruption.

References

1. Ngu, V. A., and Konstam, P. G.: Traumatic dissecting aneurysm of the abdominal aorta. Brit. J. Surg. 52: 981, 1965.
2. Tomatis, L. A., Doornbos, F. A., and Beard, J. A.: Circumferential intimal tear of aorta with complete occlusion due to blunt trauma. J. Trauma 8: 1096, 1968.
3. Rybak, J. J., and Thomford, N. R.: Acute occlusion of the infrarenal aorta from blunt trauma. Amer. J. Surg. 35: 444, 1969.
4. Hewett, R. L., and Grablowsky, O. M.: Acute traumatic dissecting aneurysms of the abdominal aorta. Ann. Surg. 171: 160, 1970.
5. David, D., and Blumenberg, R. M.: Subintimal aortic dissection with occlusion after blunt abdominal trauma. Arch. Surg. 100: 302, 1970.
6. Borja, A. R., and Lansing, A. M.: Thrombosis of the aorta caused by blunt trauma. J. Trauma 10: 499, 1970.
7. Thal, E. R., Perry, M. O., and Crighton, J.: Traumatic abdominal aortic occlusion. Southern Med. J. 64: 653, 1971.
8. Angel, R. T., and Crosthwait, R. W.: Acute traumatic dissecting aneurysm of the abdominal aorta. Texas Med. 68: 82, 1972.
9. Parmley, L. F., Mattingly, T. W., Manion, W. C., and Jahnke, E. J., Jr.: Nonpenetrating traumatic injury of the aorta. Circulation 17: 1086, 1958.
10. Malette, W. G., Armstron, R. G., and Criscuolo, D.: A second mechanism in hypotension following release of abdominal aortic clamps. Surg. Forum 14: 292, 1963.
11. Potter, D. J., and Hopkins, J. G.: Reversible paraplegia and acute renal failure due to occlusive disease of the abdominal aorta. Ann. Int. Med. 69: 777, 1968.

VETERANS ADMINISTRATION CENTER
Research "Open House"
Friday, Sept. 7, 1973
Program

9:00- 9:30 a.m.	Dr. Alton Mun, Professor of Zoology, University of Maine, Orono, "Stimulation of growth of embryonic spleen graft by homogous adult spleen in the chick embryo."
9:30-10:00 a.m.	Dr. Robert F. Ritchie, Rheumatic Diseases Laboratory, Maine Medical Center, "Plasma proteins; an untapped source of health information."
10:00-10:15 a.m.	Coffee break
10:15-10:45 a.m.	Dr. John Howland, Biology Department, Bowdoin College, "Scanning electron microscopy of the RBC in muscular dystrophy."
11:00-11:45 a.m.	Dr. Edmund Klein, Chief, Department of Dermatology, Roswell Park Memorial Institute, "Current concepts of the application of immunotherapy to neoplastic diseases."
12:30 p.m.	Luncheon, by reservation. Telephone 623-8411, extension 373.
1:30- 2:00 p.m.	Dr. Frank T. Passini, Associate Director of Alcohol Treatment Unit, Togus, "Transcerebral stimulation—biofeedback; quackery—quick cure; quandry."
2:00 p.m.	Tour of research facilities and exhibits.

Acute Alcoholic Hepatitis: Diagnosis, Treatment, Prognosis

DONNA KIEVIT THOMPSON, M.D.*

Alcoholic hepatitis is a more common disease entity than is generally appreciated. This "underdiagnosis" probably is the result of several factors: the wide spectrum of presentation of the syndrome, especially its ability to mimic other causes of acute abdomen; and, the failure on the part of physicians to think of the diagnosis, especially in dealing with patients who are not generally thought of in the alcoholic category. The purpose of this paper is to review the clinical and pathological criteria for the diagnosis of alcoholic hepatitis and to present a brief discussion of its treatment and prognosis.

Although alcoholism or problem drinking has social as well as medical facets to its definition, certain basic statements can be made as to what constitutes excessive drinking on a metabolic basis. Pathologic studies and experiments with alcohol feeding provide evidence of alcohol-induced liver injury.[27-32] The liver's metabolic limit for alcohol is 160-180 gm/day which is the equivalent of about 2/3 bottle of 86 proof whiskey per day. Excessive drinking may be defined as greater than 80 gm/day or more than 20% of total daily caloric intake derived from alcohol.[24] However, this should not lead one to believe that an absolute dose-response relationship exists between alcohol consumption and liver pathology for many other factors, most of them related to host response and as yet poorly understood, affect the end result of liver disease. Despite heavy alcohol consumption as defined above, only one in eight or 12% go on to develop cirrhosis. Seventy-five percent will have a fatty liver and 40% will have at least one bout of acute alcoholic hepatitis.

The alcoholic history of patients who present with acute alcoholic hepatitis varies widely. In one long-term study involving 169 patients, the history of heavy drinking varied from three months to thirty-six years. Although the majority could be labeled chronic alcoholics, 7% had been drinking thirty-six years. Although the majority could be labeled alcoholics, 7% had been drinking for less than one year.[7] Many of these latter patients are not obviously alcoholic, nor do many of them appear intoxicated on presentation. It is not unusual for patients to have normal blood alcohol levels when first seen with alcoholic hepatitis. Generally, however, it is possible to obtain a history of a recent marked increase in alcohol consumption, i.e., a recent "drinking spree."

The patients may be practically asymptomatic and the diagnosis made on the basis of abnormal laboratory values or biopsy, or they may present with a fulminating illness and be acutely ill. The presenting complaints, physical findings and laboratory findings may vary greatly. A number of clinical studies have yielded varying results, in part depending on the type of patient population studied, but the following gives a general picture of the frequency of presenting signs and symptoms.[1-11]

The most common presenting complaint is a triad of symptoms: anorexia, nausea and vomiting, weakness. These occur in 77% of the patients in almost all studies. Other frequent complaints are right upper quadrant abdominal pain in 46%, recent weight loss in 43% and diarrhea in 20%.

The two most frequent physical findings are hepatomegaly and jaundice. Hepatomegaly, usually with a moderately firm and extremely tender liver, is present in 81-91% of cases. Jaundice is a more variable finding, being reported at a frequency of between 37-70% of cases depending on the the series. This brings out the important point that it is very possible for alcoholic hepatitis to be present in the absence of jaundice and that one should never discard this diagnosis in an anicteric patient.[5] Other physical findings vary considerably. Fifty-five percent of patients will appear generally malnourished. Fifty percent will have extra-hepatic manifestations of liver disease such as spider nevi, gynecomastia, liver palms, etc. Splenomegaly may be found in up to 37% and ascites has been reported at a frequency varying between 35 and 73%. Twenty-two percent have some form of gastrointestinal bleedings, ranging from melena stool and coffee ground vomitus to massive hemorrhage. A temperature of greater than 100.4° is present in 56% and a source of infection is found in half of these patients. At least a mild degree of anemia is found in 70% of patients, and a more marked anemia with a hematocrit less than 30 is found in 43%. The causes of this anemia are many, including blood loss, folic acid or B_{12} deficiency, so-called dimorphic anemia, hemolysis or marrow alcohol toxicity. Leukocytosis is present in 40% with values often exceeding 40,000. The highest white count in a patient with acute alcoholic hepatitis has been recorded as 159,000.[6] Alterations of central

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nervous system function are present in 17%. These range from tremulousness through neuropathies and delirium tremens to frank coma.

The information obtained from laboratory studies can be very helpful in confirming the diagnosis of alcoholic hepatitis, but again as in the clinical presentation, results may be highly variable. Although the bilirubin is elevated in 71% of patients, it should be remembered that some may have normal values. Fractional bilirubin is invariably greater than 60% direct-reacting. Sixty-seven percent have an elevated alkaline phosphatase, but this is poorly correlated with cholestatis. The SGOT is elevated in 90%, but an important and helpful point in the diagnosis is that it is rarely greatly elevated. In 90%, the elevation is less than 500, and in 50%, less than 250. This is in marked distinction from viral hepatitis, often a difficult point in differential diagnosis. The SGPT is elevated in 50%. Invariably in alcohol hepatitis, the SGOT is more elevated than the SGPT, usually in a ratio of greater than 1.5 to 1. This ratio is probably the most important point in the laboratory diagnosis of alcoholic hepatitis for in every other form of hepatitis such as viral hepatitis the opposite is true, i.e., the SGPT is more greatly elevated than the SGOT. Not only does this SGOT:SGPT ratio help in making a differential diagnosis but it can also be helpful to sorting out patients with abnormal liver function tests who deny heavy alcohol consumption, i.e., if the SGOT:SGPT ratio is greater than 1.5 in the presence of enzyme abnormalities it is wise to be suspicious of heavy drinking and question the patient further.[1,2] The albumin is less than 3 in 60% and the gamma globulin greater than 1.7 in 72%. Hyperlipemia is present in 20% and some cases of alcoholic hepatitis have been associated with Zieve's syndrome. The BUN is greater than 20 in 21%. The prothrombin time is often elevated but this varies tremendously. As will be discussed later, the pro-time can have significance in assessing prognosis.

The differential diagnosis of acute alcoholic hepatitis may at times be difficult, by many of the problems can be avoided if the entity is especially considered and the points mentioned above carefully looked for. The two diagnoses most often confused are viral hepatitis and obstructive jaundice. Fatty liver may also be diagnosed in place of acute alcoholic hepatitis. Other diagnoses that may cause confusion are cirrhosis (not only Laennec's but other types as well), metastatic lesions of the liver, pancreatitis and peritonitis. It is especially important to determine that one is dealing with alcoholic hepatitis when surgery is being considered. Surgical procedures, as for a mistaken diagnosis of obstructive jaundice, carry an extremely high mortality rate in alcoholic hepatitis. In one study,[19] 16 of 23 patients were originally given a surgical diagnosis. Of the 10 who actually came to surgery, 7 did not survive.

One presentation of acute alcoholic hepatitis deserves special mention. Patients may present with ascites and other manifestations of portal hypertension but this is not necessarily evidence of advanced cirrhosis. Many cases have complete resolution of ascites and increased portal pressures as the acute illness subsides. This acute ascites is thought to be due to centrolobular inflammation and edema compressing sinusoids and sinusoidal filling with necrotic debris. In one study[14] of 28 patients with acute alcoholic hepatitis in whom hepatic wedge pressures were elevated and 13 of 28 had clinical manifestations of portal hypertension, none of these were found to have evidence of cirrhosis with regenerative nodules on biopsy. Thus, in the presence of acute hepatitis, ascites does not necessarily mean cirrhosis.

Often the diagnosis of acute alcoholic hepatitis can only be definitely confirmed by liver biopsy. Although the histologic picture may vary considerably, there is almost always enough characteristic pathologic alteration to make the diagnosis.[22,23] Changes are always most pronounced in centrolobular areas. There is cellular necrosis with pyknotic nuclei, ballooning degeneration and hydropic swelling. Most characteristic and pathognomonic for acute alcoholic hepatitis is hyaline degeneration, the so-called Mallory body. This hyaline material forms a fine web around the nucleus, is dense and darkly acidophilic and probably represents denatured protein, perhaps endoplasmic reticulum.[21] Although not present invariably, when seen they do establish the diagnosis. Polymorphs are seen infiltrating the sinusoids and areas of cellular debris. Varying degrees of fat or fibrosis may be seen depending on the underlying state of the liver. Frank cirrhosis with regenerative nodules and variable amounts of cholestasis and portal inflammation may also sometimes be found. Although electron microscopy is not necessary in making the pathologic diagnosis, the picture is characteristic and includes fat droplets, whorling and clumping of endoplasmic reticulum, giant and bizarre-shaped mitochondria, autophagocytic granules and dilatation of bile canaliculi with loss of microvilli.[21,30,33]

An excellent review of the treatment of acute alcoholic hepatitis may be found in GUT.[20] The basic plan is a simple one involving initially general supportive care of an often seriously ill patient. Additional aggravating problems such as bleeding or infection are searched for and treated. The cornerstone of specific therapy is abstinence. There is definitely a decrease in mortality, relapse and progression to cirrhosis with abstinence, but of course this is extremely difficult to follow through.

TABLE 1

ALCOHOLIC HEPATITIS — SUMMARY

1. Steatosis, necrosis, Mallory bodies, polymorph infiltration
2. Acute metabolic failure of the liver in serious cases
3. Results from severe drinking bout
4. Possibly complication of steatosis or cirrhosis
5. Age of patients: relatively young (30-50)
6. CLINICAL FEATURES: jaundice
   fever
   ± stigmata of cirrhosis
   ± portal hypertension (varices)
   ± coagulation defects
   ± signs impending heptic coma or hepatic coma
7. LABORATORY FEATURES: 80-90% patients: ↑ bilirubin
   ↑ BSP
   ↑ SGOT (< 250)
   SGOT > SGPT
   ↓ albumin
   40-60% patients: ↑ globulin
   ↑ prothrombin time
   leukocytosis
8. PROGNOSIS: a. risk sudden death or hepatic coma (4-30%)
   b. recovery (10-70%)
   c. risk of chronic hepatitis or cirrhosis (up to 50%)

Diet is also very important and, in the absence of encephalopathy or other signs of frank liver failure, should be high in protein. Most patients also need vitamin supplements. Very often it is extremely difficult to institute diet therapy as the patients are anorectic with nausea and vomiting. There may be accompanying gastritis or pancreatitis. Recently, artificial parenteral or supplemental diets have been tried in the early stages when eating is difficult. These include medium chain triglycerides, hyperalimentation programs and elemental diets.[20] The possible role of steroids in the treatment of acute alcoholic hepatitis has also undergone experimental investigation, but the results to date are not very promising and steroids probably have little beneficial effect outside of having some appetite-increasing value[1,2,12]

The assessment of prognosis of acute alcoholic hepatitis has been difficult. There are two basic problems: one is the variation in the groups of patients studied and the seconds is our lack of understanding of the pathogenic mechanisms involved in determining whether the final result is recovery, death or progressive cirrhosis. Studies based on biopsy groups generally contain patients who are less ill (they are well enough to have a liver biopsy) and thus the mortality is fairly low in the range of 4-8% in most series. More general groups contain sicker patients and run a mortality of from 30-75%. However, despite these difficulties, some clues of prognostic significance can be obtained.

Although no one particular complaint or physical finding has any prognostic importance by itself, generally those patients who appear overall to be more ill do poorly; however, this is not invariably true. Those patients who have evidence of preexisting liver disease also do poorly. Females seem to have more severe and more frequent attacks of alcoholic hepatitis than males and generally have a poorer prognosis, also they are affected at a younger age.

Of all laboratory studies, the prothrombin time appears to be the best prognostic indicator. The important parameter is a rising pro-time; the initial value itself is not of great importance, but if the level continues to rise or has not come down by the end of a week this is associated with a very poor prognosis. In one study, the patients could be divided into two groups on the basis of their pro-time: in the first group of 34 patients with an increasing pro-time, the mortality was 34%; in the second group of 135 patients whose pro-time did not rise in the first week although some initial values were higher than those of the first group), the mortality was 0.7%.[8] Two other findings are associated with a poor prognosis; a BUN of greater than 20 on initial evaluation which remains elevated and a bilirubin of greater than five which continues to rise after the first six days.[11]

The presence or absence of Mallory bodies on histologic examination has no prognostic significance.[16,17] nor does the degree of steatosis. The only histologic findings associated with poor prognosis are those of cirrhosis, again proving that those with underlying liver disease tend to do worse.

The one factor of treatment that appears to affect prognosis is drinking; those who abstain do better than those who keep on drinking. Follow-up liver biopsies only return to normal in those who stop drinking. The lack of efficacy of steroids has already been discussed.

Recently there have been several studies on the

natural history and outcome of acute alcoholic hepatitis. Most studies provide similar results and indicate that many patients, even those who are abstinent, go on to develop chronic hepatitis or cirrhosis. The mechanisms involved in this evolution are poorly understood and are currently under further study. The most extensive study to date on the prognosis of alcoholic hepatitis gives the following results: 169 total patients were in the study with an overall acute mortality of 30%. The mortality of that group of patients who were well enough initially to undergo liver biopsy was 4%. The five-year mortality of the survivors was 56.8% Forty-six patients were followed for twelve years and of these 11% had complete recovery. The remaining 89% had persistent hepatitis (in some cases up to twelve years) and half of these (44%) went on to develop cirrhosis.[7] Thus the risks sustained by a bout of acute alcoholic hepatitis persist for many years.

Table 1 gives a summary of the clinical, laboratory and prognostic features of acute alcoholic hepatitis.[11]

REFERENCES

1. Helman, R. A., Temko, M. H., Nye, F. W., Fallon, H. J.: "Alcholic Hepatitis — Natural History and Evaluation of Prednisolone Therapy." *Annals of Internal Medicine*, 74: 311-312, 1971.
2. Reynolds, T. B., Edmondson, H. A.: (Editorial) "Alcoholic Hepatitis." *Annals of Internal Medicine*, 74: 441-442, 1971.
3. Galambos, J. T.: "Alcoholic Hepatitis; its Therapy and Prognosis." *Progress in Liver Disease*, Vol. IV, ed. by Popper, H., Schaffer, F. New York: Grune and Stratton Inc., 1972, ch. 33.
4. Beckett, A. G., Livingstone, A. V., Hill, K. R.: "Acute Alcoholic Hepatitis." *British Medical Journal*, 2: 1113-1119, 1961.
5. Beckett, A. G., Livingstone, A. V., Hill, K. R.: "Acute Alcoholic Hepatitis without Jaundice." *British Medical Journal*, 2: 580-582, 1962.
6. Edmondson, H. A., Peters, R. L., Reynolds, T. B., et al: "Sclerosing Hyaline Necrosis of the Liver in the Chronic Alcoholic; A Recognizable Clinical Syndrome." *Annals of Internal Medicine*, 59: 646-673, 1963.
7. Lischner, M. W., Alexander, J. F., Galambos, J. T.: "Natural History of Alcoholic Hepatitis." *American Journal of Digestive Diseases*, 16:6 481-494, 1971.
8. Galambos, J. T.,: "Natural History of Alcoholic Hepatitis." *Gastroenterology*, 63:68-72, 1972.
9. Green, T., Mistilis, S., Schiff, L.: "Acute Alcoholic Hepatitis." *Arch. of Internal Medicine*, 112: 67-68, 1963.
10. Phillips, G. B., Davidson, C. S.: "Acute Hepatic Insufficiency of the Chronic Alcoholic." *Arch. of Internal Medicine*, 94: 585-603, 1954.
11. Harinasuta, U., Chomet, B., Ishak, K., Zimmerman, H. J.: "Stealonecrosis Mallory Body Type." *Medicine*, 46: 141-162, 1967.
12. Porter, H. P., Simon, F. R., et al: "Corticosteroid Therapy in Severe Alcoholic Hepatitis." *NEJM*, 284: 1350-1355, 1967.
13. Davidson, C. S.: (Editorial) "Alcoholic Hepatitis." *NEJM*, 254: 1378, 1971.
14. Reynolds, T. B., Hidemura, R., Michel, H., Peters, R.: "Portal Hypertension Without Cirrhosis in Alcoholic Liver Diseases." *Annals of Internal Medicine*, 70: 497-506, 1969.
15. Davidson, C. S., MacDonald, R. A.: "Recovery From Active Hepatic Disease of the Alcoholic." *Arch. of Internal Medicine*, 110: 592-595, 1962.
16. Rice, J. D., Yesner, R.: "The Prognostic Significance of So-Called Mallory Bodies in Portal Cirrhosis." *Arch. of Internal Medicine*, 105: 99-104, 1960.
17. Harinasuta, U., Zimmerman, H. J.: "Alcoholic Steabonecrosis; Relationship between Severity of Hepatic Disease and Presence of Mallory Bodies in the Liver." *Gastroenterology*, 60: 1036-1046, 1971.
18. Hardison, W. G., Lee, F. I.: "Prognosis in Acute Liver Disease of the Alcoholic Patient." *NEJM*, 275: 61-66, 1966.
19. Mikkelson, W. P., Turrill, F. L., Kern, W. H.: "Acute Hyaline Necrosis of the Liver; a Surgical Trap." *American Journal of Surgery*, 116: 266-272, 1958.
20. Resnick, R. H., Iber, F. L.: "Treatment of Acute Alcoholic Hepatitis." *GUT*, 13: 68-73, 1972.
21. Schaffner, F., Loebel, A. Weiner, H. A., Barku, T.: "Hepatocellular Cytoplasmic Changes in Acute Alcoholic Hepatitis." *JAMA*, 183: 343-346, 1963.
22. Porter, E. A., Bergnan, B. J., Stein, A. A.: "Acute Alcoholic Hepatitis." *American Journal of Pathology*, 46: 657-689, 1965.
23. Proceedings: 33rd Seminar of the American Society of Clinical Pathologists, Cases M9, 10, 12; Sept. 29, 1967.
24. Sherlock, S.: *Disease of the Liver*, "Alcohol and the Liver, chapter 15, pg. 412-425. Phila.: F. A., Davis Co., 1968.
25. Leiber, C. S.: "Alcohol and the Liver." *Progress in Liver Disease*, Vol. II, ed. by Popper, H., & Schaffner, F., New York: Grune and Stratton, Inc., 1965, chpt. II.
26. Galambos, J. T.: "Alcohol and Liver Disease." *American Journal of Digestive Disease*, 14: 477-490, 1969.
27. Ugarte, G., Itarriaga, H., Insunza, I.: "Some Effects of Ethanol on Normal and Pathologic Livers." *Progress in Liver Disease*, vol. II ed. by Popper, H., & Schaffner, F. New York: Grune and Stratton, Inc. 1970, chpt. 22.
28. Lieber, C. S.: "Hepatic and Metabolic Effects of Alcohol." *Gastroenterology*, 50: 119-133, 1966.
29. Klatskin, G.: "Alcohol and Its Relation to Liver Damage." *Gastroenterology*, 41: 443-451, 1961.
30. Edmondson, H. A., Peters, R. L., Frankel, H. H., et al: "The Early Stages of Liver Injury in the Alcoholic." Medicine (Baltimore) 46: 119-219, 1967.
31. Lieber, C. S., Rubin, E.: "Alcoholic Fatty Liver." *NEJM*, 250: 705-708, 1969.
32. Rubin, E., Lieber, C. S.: "Alcoholic Induced Hepatic Injury in Non-Alcoholic Volunteer." *NEJM*, 278: 869-876, 1968.
33. Rubin, E., Lieber, C. S.: "Early Fine Structural Changes in the Human Liver Induced by Alcohol."
34. Galambos, J. T., Makoto, A., Shaks, J. Z.: "The Effects of Intravenous Ethanol on Serum Enzymes in Patients with Normal or Diseased Liver." *Gastroenterology*, 44: 267-274, 1963.
35. Powell, W. J., Klatskin, G.: "Duration of Survival in Patients with Laenne's Cirrhosis." *American Journal of Medicine*, 44: 406-420, 1968.

Maine Tuberculosis and Health Association and the Maine Thoracic Society To Hold Joint Session, September 19, 1973

The Maine Tuberculosis and Health Association, and its medical advisory section, The Maine Thoracic Society, will meet in Augusta at the new Civic Center on September 19, 1973, in joint session.

Harold L. Israel, M.D., Professor of Medicine, Jefferson Medical College, Philadelphia, will give a presentation, "Controversy in Sarcoidosis" at the morning meeting of the Thoracic Society at 11:00 a.m.

The joint luncheon session will be followed by the presentation of the 1973 Roselle W. Huddilston Medal by Elizabeth J. Levinson, Ph.D., of Orono, last year's Huddilston Medalist. The Huddilston Medal Award is given annually by the association in recognition of "distinguished service and outstanding contributions in the field of health to the people of the State of Maine. . ."

The Edmund P. Wells Memorial Health Lecture will be given by Howard N. Jacobson, M.D., Harvard Medical School, Chairman, Steering Committee, New England Conference on the Quality of Life, author of many publications, and an active staff member at Boston Hospital for Women.

Sick Sinus Syndrome

A Case Report With Review of Recent Literature

LOUIS E. ROSENTHALL, M.D.*

INTRODUCTION

Sinus Bradycardia (SB) is not always benign — even in young athletic individuals. Inappropriate SB in presence of fever, exercise, CHF, Atropine or Isuprel® is distinctly abnormal and suggests a failing Sino-Atrial Node (SAN). Sick sinus syndrome (SSS) affects all ages and both sexes. It may cause crib deaths in infants and must be included (with myocardial infarction, stroke, and pulmonary embolus) in the differential diagnosis of sudden death in adults. The natural history of SSS varies with the cause of SAN failure. It may present acutely as a complication of myocardial infarction; or, it may run a chronic indolent course of 5-10 years or more as in hemochromatosis or amyloidosis. Treatment is permanent ventricular demand pacing. Prognosis without therapy is uncertain.

CASE REPORT

Mrs. D.M., a 56-year-old white female, was brought to the hospital after she suddenly passed out at home. There was no history of dizzy spells, loss of consciousness, hypertension, chest pain, angina, hemoptysis, rheumatic heart disease, thyroid disease, diabetes, stroke or transient ischemic attacks. No allergies. Medications included Lanoxin® 0.125 mg po qid; Inderal® 10 mg qid; Quinidine 400 mg qid; Regroton® tab ½ tid. Eight years prior to admission she substained an inferiolateral MI and recovered uneventfully. Eighteen months prior to admission she complained of palpitations. EKG showed atrial fibrillation with a ventricular response of 150-170 which reverted to NSR of 60-70 with digitalization. Seventeen months prior to admission EKG revealed atrial flutter with 2:1 block and ventricular rate of 130. Within hours, she reverted spontaneously to SB of 50.

On admission, she was an anxious white female with open laceration over right lateral scalp with temperature of 98.2°, Bp 130/70, resp. 12, pulse 40 with frequent irregularities. Chest was clear to P&A; heart: 1/6 non-radiating systolic murmur over apex which was palpable in 5th ICS, MCL. There were no rubs, gallops or diastolic murmurs. Neurologic and remainder of physical exam were unremarkable.

Admission EKG showed SB, multifocal PVC's; no hyperacute ST-T changes. 0.6 mg Atropine IV raised pulse from 40 to 60; lidocaine controlled her PVC's temporarily. Inderal was discontinued; Lanoxin and Quinidine were continued. On second hospital day, she complained of palpitations; frequent multifocal PVC's with runs of V-Tach were noted. IV lidocaine and Pronestyl® satisfactorily controlled ventricular instability; Lanoxin was withheld. On 5th hospital day, a nodal rhythm of 60 was competing with SB of 50. Serial enzymes were normal; serial EKG's showed sinus arrhythmia, SB and QT prolongation. Quinidine level was 8.7 mg/l (toxic: 6 mg/l). Quinidine was discontinued. SB remained the dominate rhythm. P Waves were noted to vary in comfiguration and duration. On the 12th hospital day, a ventricular demand pacer was inserted. Two days later several brief episodes of atrial fibrillation with ventricular responses of 100-120 occurred and patient was redigitalized. On the 18th hospital day, atrial flutter with 2:1 block was noted and Quinidine was added. There was no further significant atrial irritability. Her rhythm alternated between slow sinus and her ventricular pacer. Clinically she was free of syncope, palpitations or chest pain, and was discharged on the 21st hospital day on Lanoxin 0.125 mg po qid and Quinidine 400 mg po qid.

COMMENT

This case illustrated several characteristics of SSS. There was persistent SB over at least 18 months, only minimally responsive to Atropine. Several escape rhythms (see diagram) were apparent: atrial fibrillation, atrial flutter, nodal rhythm and V-tach. P waves were noted to vary in configuration and at least one EKG demonstrated sinus arrhythmia. Her fainting spell was probably precipitated by sinus arrest or V-tach: an escape rhythm that was poorly tolerated. The underlying nature of her SAN disease remains unclear. With prior MI, she could have an ischemic SAN; however, cardiomyopathy with infiltrative degeneration of the node is perhaps more compatible with the long clinical course. Escape tachyarrhythmias of atrial fibrillation and flutter occurred after, as well as before, pacemaker insertion; however, with optimal use of Lanoxin and Quinidine, it is hoped there will be controlled in the future. The pacemaker itself will protect against sinus arrest or cardiac standstill.

DISCUSSION

A sick SAN may fail intermittently in a variety of ways, as highlighted by the diagram. The diagnosis is thus based on indirect appraisals and is often difficult to establish with certainty. Ferrar[1] lists six manifestations of SAN inadequacy:

1. Persistent, severe and inappropriate SB — eg. SB in presence of exercise, CHF, fever, Atropine or Isuprel.
2. Sinus arrest for short period or sinus arrest for longer period with replacement of sinus rhythm with atrial or junctional escape rhythm.
3. Sinus arrest for longer period without escape rhythm causing cardiac arrest.
4. Chronic or intermittent atrial fibrillation secondary to permanent or intermittent SAN failure, respectively. If AV node is also inadequate, then Ventricular response to Atrial fibrillation is slow — see diagram.
5. Inability to restore NSR with cardioversion for atrial fibrillation.
6. SAN exit block not related to drug therapy.

Causes of SAN dysfunction include the following:

A. Drug induced (Vagotonia):
   Digitalis — Quinidine — Inderal —

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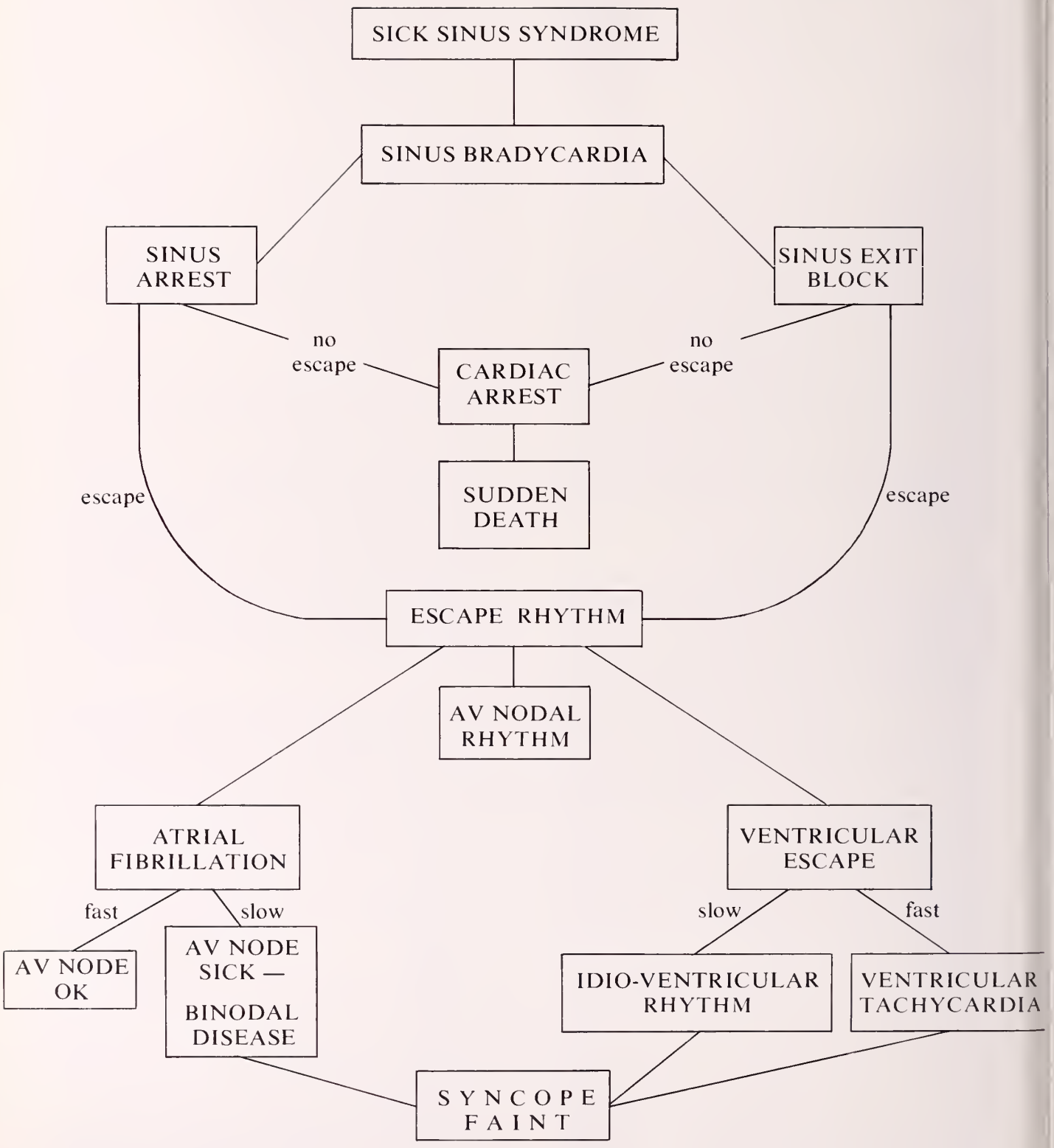
SICK SINUS SYNDROME
SINUS BRADYCARDIA
SINUS ARREST
SINUS EXIT BLOCK
no escape
no escape
CARDIAC ARREST
SUDDEN DEATH
escape
escape
ESCAPE RHYTHM
AV NODAL RHYTHM
ATRIAL FIBRILLATION
VENTRICULAR ESCAPE
fast
slow
slow
fast
AV NODE OK
AV NODE SICK — BINODAL DISEASE
IDIO-VENTRICULAR RHYTHM
VENTRICULAR TACHYCARDIA
SYNCOPE FAINT

Table 1

Excess K — Nicotine.

B. Cardiomyopathy

C. Infection/Inflammation
   Aerosol propellants with fluorinated hydrocarbons.

D. Neoplastic infiltration.

E. Traumatic — including post surgical insult to SAN.

F. Arteriosclerosis with ischemic/infarcted SAN.

G. Congenital disease:
   Fredrich's Ataxia, Muscular Dystrophy
   Familial SAN Disease without other illness.[2]

Blood is supplied to the SAN by the Right Coronary in 55% of people, and by the left circumflex in the remaining 45%.[6] SAN dysfunction complicating myocardial infarction occurs in about 5% of all acute MI's — typically with inferior or lateral infarction, in which the right or left circumflex is compromised, respectively nodal dysfunction is manifested by SB and/or transient junctional or atrial arrhythmias, particularly atrial fibrillation. In one series,[3] 31 of 32 patients with SAN disease had inferior infarctions. Conversely, SA nodal ischemia, atrial infarction and atrial arrhythmias are much less common after acute anterior septal infarction in which the left anterior descending is compromised.[8]

Monitoring a patient with suspended SSS is accomplished by out-patient 12 hour tape or preferentially inpatient telemetry. Provocative tests for SAN function include the following:

A. Treadmill — Stress
   If SB persists with exertion, syncope with loss of consciousness may ensue.

B. Atropine 1-2 mg IV
   SSS is suggested if sinus rate of 90+ is not attained

C. Isuprel 1-2 mg/min IV
   SSS is suggested if sinus rate of 90+ is not attained

D. Atrial pacing to 120-140 for 2-4 minutes: A sluggish SAN recovery time (with or without escape rhythms prior to resumption of sinus rhythm) is the most reliable indirect evidence for a failing SAN.

The electrocardiogram may show abnormally wide P waves of intra-atrial block, or P waves changing their shape, direction or vector suggesting new sites of automaturity outside the failing SAN. Wandering atrial pacemaker usually paces the ventricles faster than the underlying SB and may be regarded as an escape rhythm. Varying degrees of AV block or Bundle Branch Block may coexist with SSS indicating defective AV node or Purkinje tissue, respectively.

Clinical manifestations of SSS are multifaceted and often intermittent. The common pathophysiology is that of hypoperfusion of vital organs — especially brain, lung and heart. Symptoms include transient ischemic attacks, disordered gait or balance if the vertebro-basilar circulation is involved; or, visual disturbances, sensorimotor impairment if the carotids are affected. Acute pulmonary edema may be precipitated by low cardiac output from ineffective escape tachyarrhythmia, sinus arrest, or even persistent severe SB. If the coronaries are under-perfused by these same mechanisms, syncope, loss of consciousness, or even cardiac arrest may occur.

SSS is most common in the older patient, often with coexistent arteriosclerotic coronary artery disease; however, SSS may occur at any age in either sex. James[4] reported a young athlete in whom vague symptoms began at age 13 when a relatively slow minus rate went unappreciated. At 14, his resting pulse was 42 and rose only to 70 with exercise. By 15½ he blacked out several times and at 18 he suddenly dropped dead during a football game.

Once the diagnosis of SSS is evident, treatment of choice is permanent ventricular demand pacing. Should a patient refuse pacing, and since chronic Atropine or Isuprel therapy is difficult, oral ephedrine can be tried.

Summary

In conclusion, SB is a common early manifestation of SSS and the former should never be assumed to be benign even in young asymptomatic individuals. Inappropriate SB — in the presence of fever, exercise, CHF, Atropine or Insuprel — is particularly suggestive of SSS. The natural history varies from several days to several years, depending on the cause of SAN failure. Symptoms include syncope, CHF, and transient ischemic attacks. Once recognized, permanent ventricular demand pacing is indicated. Prognosis without treatment is uncertain.

References

1. Ferrer, M.I.: The Sick Sinus Syndrome; Circulation 47:635-641, 1973.
2. Spellberg, R.D.: Familial Sinus Node Disease; Chest 60:246, 1971.
3. Rokseth, R., et al: Sinus Arrest in Acute Myocardial Infarction; Brit. Heart Journal 33:639, 1971.
4. James, T.N., et al: Sudden Death in Young Athletes; Ann. Int. Med. 67:1013, 1967.
5. James, T.N.: Anatomy of the Human Sinus Node; Anat. Rec. 141:109, 1961.
6. James, T.N.: Pulse and Impulse in the Sinus Node; Henry Ford Hosp. Med. J. 15:275, 1967.
7. Ferrer, M.I.: The Sick Sinus Syndrome in Atrial Disease; JAMA 206:645, 1968.

Hypertrophic Pyloric Stenosis in the Adult

Case Report and Review of the Literature

PAYSON S. ADAMS, JR., M.D.*

This entity was first described in infants by Fabricius Hildanus in 1627. The first adult case was reported by Jean Cruveilhier in 1835. Ages range from 32-82 according to Seaman.[11] Knight[7] found, in his series, a range of 37-66 years. Christiansen and Grantham[2] found the range between 14 and 85 and Heibert and Farris' series was 31-71 years.[5] The average age was 47.5 for Christiansen and Grantham[2] and 50.1 for Knight.[7] The duration of symptoms was variable; Hiebert and Farris[5] found a range of 6 months to 30 years with an average of 8 years. Christiansen and Grantham[2] discovered an average duration of 12 years. As in infantile stenosis there is generally a predilection for males. Kirklin and Harris[6] found approximately 4:1. Craver[3] had a greater disparity with 10:1. Hiebert and Farris[5] series contradict this with 64% female while Christiansen and Grantham[2] found a 1:1 ratio. According to Hakhamimi and Coghill,[4] who reviewed the literature in 1968, the incidence was 1 in 106 to 1 in 2000 upper gastrointestinal examinations. At that time, there were 156 cases in the literature with the greatest percentage appearing since 1950.

Case Report

An 86-year-old Cuban male admitted for repair of a right inguinal hernia had a long history of epigastric distress, especially after eating. He reported that since 1959 when Castro came to power and the food became almost intolerable he had had difficulty with mild pain and bloating in the upper abdomen. He indicated that he had projectile vomiting which occurred after eating and would persist for up to 3 days and then subside for variable lengths of time, usually 3 days. He had no history of weight loss. He had not previously had symptoms of ulcer disease and had always been healthy. He stated that no one in his family had had, or was operated on for similar problems. The physical examination was negative with the exception of a right inguinal hernia. Upper gastrointestinal examination revealed a narrowing and elongation of the pylorus without gastric obstruction which was suggestive of a neoplastic process, but hypertrophic pyloric stenosis was considered. A bone scan, metastatic series, and a gastrocamera study was done and found to be negative. A laparotomy was performed, the pylorus was palpated revealing obstruction to the stomach outlet. A Heineke-Mikulicz pyloroplasty and vagotomy were done and the patient was discharged improved.

Classification and Etiology

Skoryna[12] has suggested classifying the disease into:

A. Primary: (1) Focal; (2) Diffuse, (a) With proximal gastric lesions; (b) Without proximal gastric lesions.

B. Secondary associated with a distal obstructive lesion.

The etiology is obscure but it is generally believed to be congenital with persistence from infancy. Horton has shown that in the normal fetus a thick pylorus is found at 5-6 months and this is postulated to persist. It is thought to be precipitated by spasm. It has been suggested that extragastric disease such as chronic cholecystitis, appendicitis, or nervousness might cause the spasm. Allergy, hyperadrenalism and vitamin deficiencies have been incriminated. Some authors have suggested that it's due either to a preponderance of sympathetic influences or impaired or tardy development of vagal inhibition. Others see it as an ancestral throwback to types in which the gizzard is located in the same spot. Belding and Kernohan[1] found degenerative changes in the ganglia cells and suggest this as the cause. Inheritance has always been considered because of the predominance of male over female and there have been some reports in which father and son have had the disease. Psychoneurotic tendencies have been pointed out by McCann and Dean.[9]

Anatomy and Pathology

The normal anatomy of the pylorus consists of two muscle layers, the inner circular and outer longitudinal, 50% of the latter interlace with the circular. Contraction of the longitudinal layer causes an increase in the diameter of the pylorus. The circular fibers have two bands, the proximal and distal which abut against each other on the lesser curvature in what is called the muscle torus or knot. The normal thickness is 3-8 mm with an average of 4.4 mm (Craver).[3] Others have found the range of 3.8-8.5 mm with an average of 5.8 mm. Knight[7] measured a range of 4.7 mm with an average of 5.1 mm. A normal canal length according to Seaman[11] is 5-10 mm.

In the pathological condition, grossly the diffuse form is a bulbous or fusiform tumor firm and rubbery with its greatest diameter on the duodenal side tapering proximally into the antrum. The wall thickness may range from 9-15 mm with an average of 11.2 mm (Seaman).[11] Craver[3] found a range of 12 mm-20 mm with an average of 15.4 mm and Knight[7] had a range of 10-15 mm. According to

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Craver,[3] microscopically there is hypertrophy and hyperplasia of the circular muscles with inflammatory changes and fibrosis. These findings have also been seen occasionally in the longitudinal fibers. Belding and Kernohan[1] found degenerative changes in the myenteric plexes or pyloric ganglia cells. Ectopic smooth muscles in the gastric mucosa have been found and shown to be hypertrophied.

A focal type of involvement has been seen which involves primarily the torus although it has been described on the greater curvature. This has not been noted in infants. There seems to be less predilection for males in this type according to Seaman.[11] It may represent an early diffuse type (Skoryna).[12]

In either type, gastric ulcers may be seen as often as 66% of cases. Gastritis is seen in 55% of the cases according to Seaman.[11] These findings, it has been suggested, may be produced by the stenosis and consequent retention, dilitation or general antral stimulation. These in turn may be responsible for the spasm and subsequent hypertrophy. Gastric carcinoma is rarely seen.

Symptoms

The signs and symptoms may persist from infancy and may vary from 1-42 years according to Craver.[3] There may be no symptoms in the face of radiologic evidence. There are no characteristic symptoms but some may include epigastric pain worsened with food and not relieved with antacids. Often there is frequent vomiting, especially after eating. Weight loss is seen. Eructation, dyspepsia, a lump in the stomach or fullness is mentioned. Other symptoms are anorexia and early satiety. Quite often there is no night pain. When a gastric ulcer is present then signs and symptoms are of this disease.

Diagnosis

The diagnosis is basically radiological until actual histological sections can be made. Gastric analysis or other laboratory aids are of no value. X-ray examination shows elongation and narrowing of the pyloric canal. Often a cleft or niche in the mid-portion corresponding to the muscle torus is seen but this does not represent an ulcer. According to Seaman,[11] this is the most diagnostic sign but not often present. Kirklin and Harris[6] found a concavity in the base of the duodenal bulb as helpful but it is also often absent. Evidence of flexibility is seen and linear folds of mucosa in the canal are helpful. The canal may be curved, hourglassed or straight. Gastric emptying is usually normal and thus usually no retention. Hyperparastalsis is occasionally seen, usually when other pathology is present. Kirklin and Harris[6] also state that eccentric placement of the canal closer to the lesser curvature is seen; the antral mucosa proximal to the tumor is wrinkled and contains deep transverse sulci.

Fig. 1. Note elongated pyloric canal with linear mucosal folds. Indentation in the base of the duodenal bulb is present.

Fig. 2. A niche in the mid portion of the canal represents the muscle torus. There are deep transverse sulci in the antrum and an indentation into the base of the bulb. Figure 1 and 2 compared reveals some flexibility of the pyloric canal.

The differential possibilities include gastric carcinoma, antral spasm, prepyloric ulcer with or without cicatrization, encircling myoma and perigastric adhesions. Pancreatic anomalies or tumor should be considered as well.

Treatment

Most authors agree that the treatment is surgical as antispasmatics are of no value. Many types of procedures have been done. It is generally felt the best is a Billroth I with partial gastrectomy. According to Knight,[7] this procedure removes the pathology and allows histologic study of the pylorus to rule out malignancy. It's quick and easy, more

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The Diagnosis and Management of the Anemia Associated with Cardiac Prosthetic Valves

ROGER LANDRY, M.D.*

The association of anemia and cardiac prosthetic devices, first documented by Hufnagel et al,[1] in 1954, is a clinical entity encountered with increasing frequency since the advent of the widespread use of these devices in the treatment of many types of heart disease. Stohlman in 1956,[2] Brodeur in 1965,[3] and Nevaril in 1968,[4] all substantiated the hypothesis that the mechanism of this anemia is intravascular hemolysis secondary to mechanical trauma to the red blood cells. The trauma is thought to be caused by turbulence induced by the prosthesis resulting in shearing forces sufficient to fragment red cells. Therefore, any condition which results in increased turbulence, i.e., paravalvular leaks,[5] increased cardiac output,[6] ball valve variance,[7] or valve type and size,[8,9,10] causes more significant hemolysis and is more likely to result in anemia.

The incidence of hemolysis is extremely difficult to assess because of the many variables to be considered in any one study. The type, location, and size of the prosthetic valve, the time of the study after surgery, the number of prosthetic valves, and whether the valve is functioning properly, are all examples of these variables. Incidences of hemolysis have been reported in the range of 68-100%[3,10] for aortic prosthetic valves. Specific incidences for mitral valves are more difficult to come by, but a large series,[11] quantitating hemolysis in all types of prosthetic valves, gave an overall incidence of 64%, suggesting the incidence of hemolysis for mitral valve prostheses is much less than that of aortic valves. A recently reported series noted an incidence of 15% for hemolytic anemia with aortic prostheses,[10] while another gave incidences of between 5-10%. These studies suggest that a state of compensated hemolysis must exist in many patients with aortic valve prostheses.

CASE REPORTS

Case 1 — C. M., a 25-year-old housewife, was admitted to the hospital for evaluation of an anemia which had been progressive over the last 11 months. She had a long history of rheumatic heart disease and, two years previously, had undergone aortic, mital, and tricuspid valve replacement for severe triple valve disease with congestive heart failure. Following surgery, she had returned to normal exercise tolerance, although she was maintained on a regimen of digoxin and diuretics. Because of mild anemia one year previously, iron, in the form of ferrous sulfate in the dihydrate salt (Feosol® Spansules) and folic acid were added to her regimen. Her hematocrit dropped 8% during the seven months prior to admission, while on a well balanced diet and without a history of blood loss. Physical examination revealed normal valve sounds, a grade II mid systolic murmur at the apex, a grade III ejection systolic murmur over the right sternal border, and no diastolic murmurs. Laboratory data revealed a hemoglobin of 8.2 gm%, hematocrit 25%, reticulocyte count 9.8%, platelets 433,000, and a peripheral smear showing marked anisocytosis, poikilocytosis, numberous fragmented red blood cells, and hypochromia. The bilirubin was 1.6 mgs%, 0.2 direct and 1.4 indirect, serum iron 42, iron binding capacity 372, and plasma hemoglobin 50 mgs%. The Coombs test, direct and indirect, and tests for fibrin split products were negative. Fibrinogen and prothrombin time were normal. Stools for occult blood were negative. The LDH was 1220, the SGOT 105, the urine was strongly positive for hemosiderin and faintly positive for hemoglobin. Fluoroscopy revealed normally functioning prosthetic poppets. Bone marrow examination demonstrated erythroid hyperplasia with absent iron stores.

The patient was given 2 units of packed red blood cells, and started on 300 mg of ferrous sulfate tid and folic acid 1 mg daily. She was discharged with a hematocrit of 33% and a 5% rise was noted over the next 2 months.

Case 2 — J. T. is a 54-year-old white female housewife with a long history of rheumatic heart disease with mitral stenosis and mitral insufficiency documented in April of 1966 by cardiac catheterization. She underwent replacement of her mitral valve with a Starr-Edwards prosthetic valve in December of 1966, and was discharged on iron therapy (Feosol Spansules, one daily) in addition to Lanoxin® and diuretics. Before discharge, she was noted to have an apical pansystolic murmur with radiation to the axilla. In late 1968, she was found to be anemic with a hemoglobin of 8.6 gms% and a hematocrit of 32%. Further testing revealed a peripheral smear showing hypochromia with numerous fragmented red blood cells, reticulocyte count of 9.4%, serum iron 24, iron binding capacity 609, LDH 2280, and SGOT 30. Bone marrow was interpreted to be normal except for increased erythropoiesis and absent iron stores. The Coombs test, direct and indirect, was negative, plasma hemoglobin 8.3 mgs%, serum haptoglobin absent, stool guarac — negative. Urine hemoglobin was 4.1 mgs/24 on one occasion, with a repeat determination showing an overnight value of zero and a daytime value of 0.3 mgs%. The patient was transfused with packed red blood cells and discharged on Folic Acid 5 mg bid and Ferrous Sulfate 300 mg tid.

She did well and in April of 1970 she had a hemoglobin of 12.3 gms%, a hematocrit of 36%, an LDH of 1260, an SGOT of 120, and a reticulocyte count of 2%. Cardiac catheterization at that time revealed severe mitral insufficiency and she subsequently underwent correction of the paraprosthetic valve leak in June 1970. Her apical murmur persisted and another catheterization in June of 1971 revealed mild-moderate mitral insufficiency. Hemoglobin was 14.8 gms%, hematocrit 42%, and LDH 485. She has maintained on Ferrous Sulfate 300 mg tid.

DISCUSSION

Clinical Picture: The clinical presentation of the anemia associated with prosthetic heart valves is

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*Intern, Maine Medical Center, Portland, Maine 04102.

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Indications: For the treatment of trichomoniasis in both male and female patients and in the sexual partners of patients with a recurrence of the infection provided trichomonads have been demonstrated by wet smear or culture. The oral tablets are indicated also for acute intestinal amebiasis (amebic dysentery) and amebic liver abscess.

Contraindications: Evidence or history of blood dyscrasia, active organic disease of the CNS, the first trimester of pregnancy and a history of hypersensitivity to metronidazole.

Warnings: Use with discretion during the second and third trimesters of pregnancy and restrict to those pregnant patients not cured by topical measures. Flagyl (metronidazole) is secreted in the breast milk of nursing mothers. It is not known whether this can be injurious to the newborn.

Precautions: Mild leukopenia has been reported during Flagyl use; total and differential leukocyte counts are recommended before and after treatment with the drug, especially if a second course is necessary. Avoid alcoholic beverages during Flagyl therapy because abdominal cramps, vomiting and flushing may occur. Discontinue Flagyl promptly if abnormal neurologic signs occur. Exacerbation of moniliasis may occur. In amebic liver abscess, aspirate pus during metronidazole therapy.

Adverse Reactions: Nausea, headache, anorexia, vomiting, diarrhea, epigastric distress, abdominal cramping, constipation, a metallic, sharp and unpleasant taste, furry or sore tongue, glossitis and stomatitis possibly associated with a sudden overgrowth of *Monilia*, exacerbation of vaginal moniliasis, an occasional reversible moderate leukopenia, dizziness, vertigo, incoordination and ataxia, numbness or paresthesia of an extremity, fleeting joint pains, confusion, irritability, depression, insomnia, mild erythematous eruptions, "weakness," urticaria, flushing, dryness of the mouth, vagina or vulva, pruritus, dysuria, cystitis, a sense of pelvic pressure, dyspareunia, fever, polyuria, incontinence, decrease of libido, nasal congestion, proctitis, pyuria and darkened urine have occurred in patients receiving the drug. Patients receiving Flagyl may experience abdominal distress, nausea, vomiting or headache if alcoholic beverages are consumed. The taste of alcoholic beverages may also be modified. Flattening of the T wave may be seen in ECG tracings.

Dosage and Administration: For Trichomoniasis. *In the female:* One 250-mg. tablet orally three times daily for ten days. Courses may be repeated if required in especially stubborn cases; in such patients an interval of four to six weeks between courses and total and differential leukocyte counts before, during, and after treatment are recommended. Vaginal inserts of 500 mg. are available for use, particularly in stubborn cases. *When the vaginal inserts are used,* one 500-mg. insert is placed high

in the vaginal vault each day for ten days and the oral dosage is reduced to two 250-mg. tablets daily during the ten-day course of treatment. Do not use the vaginal inserts as the sole form of therapy. *In the male:* Prescribe Flagyl only when trichomonads are demonstrated in the urogenital tract, one 250-mg. tablet two times daily for ten days. Flagyl should be taken by both partners over the same ten-day period when it is prescribed for the male in conjunction with the treatment of his female partner.

For Amebiasis. *Adults:* For acute intestinal amebiasis, 750 mg. orally three times daily for 5 to 10 days. For amebic liver abscess, 500 to 750 mg. orally three times daily for 5 to 10 days. *Children:* 35 to 50 mg./kg. of body weight/24 hours, divided into three doses, orally for ten days.

Dosage forms: Oral tablets 250 mg.
Vaginal inserts 500 mg.

References:

1. Perl, G., and Ragazzoni, H.: Flagyl in Treatment of "Trichomonas Vaginalis" Vaginitis, Obstet. Gynecol. *19*:595-598 (May) 1962. **2.** Kean, B. H.: Trichomoniasis in Males (Letters to the Journal), J. A. M. A. *186*:273 (Oct. 19) 1963. **3.** King, A. J.: Current Therapeutics: CLVI.—Metronidazole in the Treatment of Trichomonal Infections, Practitioner *185*:808-812 (Dec.) 1960. **4.** Watt, L., and Jennison, R. F.: Clinical Evaluation of Metronidazole: A New Systemic Trichomonacide, Br. Med. J. *2*:902-905 (Sept. 24) 1960. **5.** Watt, L., and Jennison, R. F.: Metronidazole Treatment of Trichomoniasis in the Female, Br. Med. J. *1*:276-279 (Feb. 3) 1962. **6.** Teton, J. B., and Treadwell, N. C.: Evaluation of a Systemic Trichomonacide, Obstet. Gynecol. *21*:356-362 (March) 1963. **7.** Durel, P.; Roiron, V.; Siboulet, A., and Borel, L. J.: Systemic Treatment of Human Trichomoniasis with a Derivative of Nitro-Imidazole, 8823 R. P., Br. J. Vener. Dis. *36*:21-26 (March) 1960. **8.** Bertrand, P., and Leulier, J.: Essais cliniques sur la trichomonase des partenaires des femmes infestees (Proceedings of the 1st Canadian Symposium on Non-Gonococcal Urethritis and Human Trichomoniasis, Montreal, 1959), Gynaecologia *149*:93-96 (Suppl.) 1960. **9.** Poole-Wilson, D. S.: The Diagnosis and Management of Chronic Infection of the Bladder, Practitioner *186*:429-437 (April) 1961.

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well demonstrated in these patients. The familiar signs and symptoms of anemia (tachycardia, dyspnea on exertion, pallor, and easy fatigability) are seen, the intensity of which is dependent on the degree of anemia. The signs and symptoms of iron deficiency anemia are seen on rare occasion. (Cheilosis, central loss of tongue papilla, brittle spoon nails, loss of head hair, esophageal web, gastric atrophy and menorrhagia.) Hepatosplenomegaly is also an inconstant finding. The peripheral smear reveal either normochromic, normocytic red blood cells or slight macrocytosis early in the process, but invariably, after 3-4 months post-op, microcytosis and hypochromia are noted. Platelets and leukocytes are normal except for occasional hypersegmented polymorphonuclear leukocytes when there is associated foliate deficiency. The characteristic fragmented red blood cells (helmet, burr cells) are invariably seen. Anisocytosis, poikilocytosis, and polychromatophilia are also noted as well as a reticulocytosis of 3-25%, depending on the degree of hemolysis.

The characteristics of iron deficiency anemia; namely, low serum iron, elevated total iron binding capacity, and a marrow devoid of iron are invariably found. The total bilirubin may be slightly elevated with a much larger proportion being unconjugated (indirect) bilirubin. The Coombs test is usually negative and, if positive, should suggest another cause of hemolytic anemia. The lactic dehydrogenase, specifically fractions 1 and 2 (RBC) is elevated, as is the serum glutamic-oxalacetic transaminase, although to a much smaller degree. Haptoglobin is low or entirely absent; hemopexin is low. Increased plasma free hemoglobin may be found, as well as hemoglobinuria, hemosiderinuria and increased iron excretion from — 2.2 — 14.6 mg/24 hours (normal < 1 mg/24 hours). These values are features of this anemia as well as any condition with intravascular hemolysis. As would be expected, the red blood cell survival is significantly reduced.

Pathogenesis

The mechanism of this anemia, then, obviously involves intravascular hemolysis. An intrinsic red cell defect is unlikely because red cells from afflicted patients have a normal survival time when transfused to patients without valve prostheses.[3] The negative Coombs test makes the presence of anti-erythrocyte antibodies unlikely. The extracorpuscular factor in these cases, as mentioned previously, is the turbulence created by the prosthetic valve. Turbulence is created by a valve gradient and is also related to valve type and size, the presence of paravalvular leaks, and an increased cardiac output for any reason, including anemia.

Turbulence causes red blood cell fragmentation and intravascular hemolysis. The hemoglobin thus released into the vascular space has several possible fates:[12,13]

1) It can be immediately bound to haptoglobin and this hemoglobin-haptoglobin complex removed by the reticulo-endothelial system where it is subsequently degraded to bilirubin.
2) The hemoglobin may dissociate into A and B dimers, be filtered in the kidney, and reformed or absorbed as dimers by the proximal tubules. Here it is metabolized to hemosiderin and ferritin. The cells are subsequently desquamated, and it is then that hemosiderin is found in the urine. This process takes approximately 4 weeks, and hence, urinary hemosiderin is never found prior to 4 weeks post-operatively.
3) When hemolysis is severe, hemoglobin may be directly oxidized to methemoglobin which then releases its heme group to albumin to form methemalbumin. Methemalbumin is cleared from the circulation, presumably by the reticuloendothelial system and by hemopexin, which has a higher affinity for heme than albumin. The heme found to hemopexin is transported to the liver parenchymal cells where it is converted to bilirubin. Many hypothesize that the albumin merely binds the heme until hemopexin is made available. Indeed, since hemopexin is not an acute phase reactant as is haptoglobin, it is considered to be a better indicator of hemolysis.[17,12]
4) With severe hemolysis the dimers of hemoglobin are filtered at a rate much geater than the reabsorptive rate of the tubular cells. Hemoglobinuria then occurs.[13]

Clinical Assessment of Hemolysis

A method of quantitating the degree of hemolysis would certainly be of help in the management of cardiac hemolysis by helping to determine those patients who would benefit from replacement of the prosthetic valve, as well as those most likely to develop anemia. Eyster[10] has proposed a system of evaluation of cardiac hemolysis based on a number of parameters. She suggests that haptoglobin levels be obtained during the first 3 to 6 months following surgery. Virtually all patients with cardiac prosthetic valves have decreased haptoglobin levels, but the absence of haptoglobin denotes significant degrees of hemolysis. In patients with the absence of haptoglobin, schistocyte counts, reticulocyte counts, lactic dehydrogenesase and bilirubin determinations should be obtained at frequent intervals of 3-6 months. Examination of urine for hemosiderin should be routinely done at 6 months intervals. The criteria for assessment of cardiac hemolysis are as fol-

lows:

Mild hemolysis — Hemosiderinuria or absence of haptoglobin, with schistocyte count (5) of <10/1,000 RBC, reticulocyte count (R) < 5% and lactic dehydrogenase (LDH) < 500.

Moderate — Hemosiderinuria or absence of haptoglobin, S > 10/1,000, R > 5%, LDH > 500.

Severe — All of the criteria for moderate hemolysis, plus hemoglobinuria.

Myhre[14] has demonstrated a corrolation between the red blood survival time and the lactic dehydrogenase, in the absence of other conditions causing elevated lactic dehydrogenase

200 or less	=	normal RBC survival
500	=	X2 RBC destruction
750	=	X3 RBC destruction
1000	=	X4 RBC destruction

Of the many parameters used to evaluate hemolysis, none are without the potential for misinterpretation.

The red blood cell survival is, of course, the most specific but it is long and expensive. LDH is an excellent parameter but only in the absence of other conditions with elevated LDH. Haptoglobin is an excellent indicator but it is an acute phase reactant. Hemopexin is reliable, but not available in all areas.

Treatment

Unless there is obvious valve malfunction with a significant hemodynamic abnormality, medical therapy is the preferred initial approach to cardiac hemolysis. Therapy should be instituted immediately upon the discovery of hemosiderinuria, since it is usually persistent and will eventually lead to iron depletion. Therapy should continue indefinitely. It should be stressed that anemia need not be present before beginning treatment because one can have significant hemolysis and manage to maintain a normal hematocrit by increasing red blood cell production up to eight fold.

Medical Treatment Consists of:

1) Limitation of activities, since exercise increases the cardiac output with resulting increased turbulence and subsequent hemolysis.[6]
2) Transfusions of packed red blood cells when anemia develops in order to decrease cardiac output.
3) Folic acid 1 mg/day since the increased turnover of red blood cells increases the requirements of folic acid, and
4) Most importantly, *iron therapy*.

In the normal individual, iron absorption equals excretion at approximately 1 mg/day. In the iron deficient state, approximately 5 mg is absorbed from the diet. If oral iron therapy is instituted up to 30-40 mg may be absorbed each day, an amount that will decrease as the iron deficient state is corrected.[15,16]

Iron is absorbed in the duodenum and upper jejunum. Various "promoters" of iron absorption, such as ascorbic acid, succinic acid, or sorbitol, are indeed promoters of absorption but are fraught with numerous side effect, and are therefore not practical. There are numerous preparations available which are "long acting" or have "delayed absorption." They are designed to reduce the side effects of iron therapy. These preparations do indeed have fewer side effects because of the fact that little iron is actually absorbed. Any iron not absorbed by the time the preparation reached the mid jejunum will not be absorbed. Ferrous Sulfate 300 mg tablets, each containing 60 mg of elemental iron, is the best preparation available. Doses of at least 300 mg tid are necessary to replenish the urine iron losses and maintain the increased production of red blood cells. Ferrous Sulfate should be taken on an empty stomach to facilitate maximal absorption.

Side effects of treatment consist of constipation, diarrhea, nausea, and, occasionally, heartburn. If symptoms develop, the dose may be reduced by one-third or one-half, or the iron may be taken with meals. Another alternative is to take a fourth dose late in the evening.

If oral iron is not adequate, or not tolerated by the patient, the cautious administration of parenteral iron should be attempted. Although rare, the anaphylactoid reactions occasionally seen with iron therapy, make this route of administration less preferred. Therapy must be indefinite and probably life long.

Only when anemia persists, in spite of adequate medical therapy, should surgery be considered. Hemolytic anemia, unresponsive to the above regimen usually involves significant hemodynamic alterations, and, therefore, warrant surgery.

References

1. Rose, J. C., Hufnagel, C. A., Fries, E. D., Harvey, W. P., and Partenope, E. A.: The hemodynamic alterations produced by a plastic valvular prosthesis for severe aortic insufficiency in man. J. Clin. Invest. 33: 891, 1954.
2. Stohlman, F., Jr., Sarnoff, S. J., Case, R. B., and Ness, A. T.: Hemolytic syndrome following the insertion of a lucite ball valve prosthesis into the cardiovascular system. Circulation 13: 586, 1956.
3. Brodeur, M. T. H., Sutherland, D. W., et al: Red blood cell survival in patients with aortic valvular disease and ball valve prosthesis. Circulation 32: 570, 1965.
4. Nevaril, C. G., Lynch, E. C., Alfrey, C. P., and Hellums, J. D.: Erythrocyte damage and destruction induced by shearing stress. J. Lab. Clin. Med. 71: 784, 1968.
5. Rodgers, B. M., Sabiston, D. C., Jr.: Hemolytic anemia following prosthetic valve replacement. Circulation. Supplement 1 to vols. 39 and 40: 1-155, 1969.

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President's Address*

Reflections of a Middle-Aged Idealist

GEORGE W. WOOD, III, M.D.

These remarks might best be titled — Reflections of a Middle-Aged Idealist, voiced on an evening marking the completion of a score of years of political experience — euphamistically called "Public Life." The reflections will be considered thoughts, not imputations. Middle age is easily defined — it begins the year you realize there is not enough time left to do all the things you planned and wanted to do. My idealism is, I would like to believe, the pursuit of high and noble principles and not a sign of a visionary or impractical person.

The twenty years began in County Society meetings when a few young Turks opined — twenty-five years for an individual to be Secretary of the Society was a bit much. Eager, yea outspoken, and unschooled, our efforts were rebuffed. However, the following year, change occurred, and and in a more kindly fashion.

Lessons No. 1 and 2: Be prepared and be kind.

An early contact with the State Association occurred in a few years when our community achieved the dubious distinction of having a physician whose patients enjoyed the highest incidence of serum hepatitis in the country. With the cooperation of many, a detailed study was completed. The physician appeared and agreed to change his grandmother's ways. For it was grandmother who sterilized the equipment. He was removed from the State and County organizations, but continued practice until his death.

Lesson No. 3: Develop more effective means of self-discipline, for medical freedom is the luxury of self-discipline.

Events of the recent past suggest we are recognizing this, a fact long accepted by the oft maligned American Medical Association. My eight years in the American Medical Association House of Delegates allowed me to watch, and occasionally to participate in the transformation of an organization personified in earlier times by its President speaking eloquently to an empty Madison Square Garden in defense of its position — to one which has exercised leadership in many areas. Since 1965, positions espoused by your Delegate, have, at the particular moment, more often been rejected than accepted, but time has given pleasing evidence of new directions developed by the AMA.

Lessons No. 4 and 5: The American Medical Association is becoming a progressive spokesman for medicine, and change takes time. During this same eight years, I have served on the Maine Medical Association Executive Committee. The other day, I read the minutes of some of the 1950's meetings. One sentence most clearly described the difference between 1953 and 1973. A member asked, seemingly with a sigh, when the meeting would adjourn, for it started at 2:30 of a Sunday afternoon, and the hour was growing late — it was 4 o'clock. That era is past. The members gather at 10 — leave at 5:30 — feeling there is still much to do. The drinks and lovely dinner are no longer part of the scene. We have tried to anticipate the future, rather difficult when we are just getting comfortable with the present, but through the years, there has been the emergence of selected alternatives, such as the Maine Medical Education Foundation, Emergency Medical Programs, Peer Review, Physicians Assistants, Continuing Education, renewed interest in the Medical Education in Maine, expanded legislative functions . . . and in the last 3 years, a rapidly growing membership. This membership has restructured the Association, voted a dues increase, and now wisely presses for an expanded administrative staff to meet the broadening demands. These demands can best be illustrated by two examples.

Because of certain factors, notably an able lobbyist, legislation has assumed a more prominent role for several months. We have done battle, most times quietly, but sometimes with much clanging of swords, in order to persuade the legislators to our view. In this, the Woman's Auxiliary has been constructively involved. Though measurements of success are imprecise — I think we have earned a run around the track come the finish of this legislative race. The other area not permitting a parsimonious portion of physician participation is Peer Review. Capturing hours of labor from one member has meant more work for others.

Lesson No. 6: Able additions to staff increase demands on membership.

Since October 1972, I have had the pleasure of stumping the State, speaking at all County Societies — except noble Piscataquis — a grievous omission. This spring, the Board of Directors of the Woman's Auxiliary, and your President, met — a step in the formation of a

*Presented at the annual session of the Maine Medical Association, June 18, 1973.

stronger alliance. In the Eastern Maine region, we have given the public increased exposure to the Maine Medical Association programs. The legislators are increasingly aware of our positions. These, and an array of other contacts and meetings, have had me in rather constant motion — constructive — I hope not brownian.

The problems have been of varying vintage. A common public refrain is the physician shortage, particularly in rural areas. Is this new? — hardly. Voltaire in the early 1700's observed, "Physicians cling to cities: there are few of them in country places. Cities contain rich citizens; their maladies spring from debauchery, gluttony and excitements." A 1973 version — Portland, yes — Island Falls, no. There are, however, many other issues to be joined. A beginning to their resolution is the development of our talent to recognize and use the talents of others, one aspect of leadership. Our leadership must be bred in an atmosphere hospitable to divergent ideas, one which rids itself of a passionate penchant for the past, recognizing the social policy of the 1970's is not that described as ideal by Lord McCauley, some 120 years ago, nor that which was in existence as recently as 1930. A social policy obdurate in its position that the Federal Government's role was to keep the peace and umpire. Realizing this is now illusory, we must *not,* however, accede to the notion government is responsible for treating every personal and social ill, a position which would solve problems by throwing money at them.

With confidence based on knowledge, experience and deep concern for people, we should become the articulate advocate of selected alternatives for the future. Quoting Mr. Justice Holmes, "I find the great thing in this world is not so much where we stand, as in what direction we are moving . . . we must sail, sometimes with the wind and sometimes against it . . . but we must sail and not drift nor lie at anchor." This is stated more harshly and in the idiom of exaggeration by Toffler in "Future Shock." Read it if only to rouse you to thought in order to rebut or refute, but read it, for in the morass of words, there is a theme undeniable. The theme is simple and one which you have long acknowledged. *Change is unavoidable.* A solution to change is you and me, not our surrogate, deeply involved in determining the rate and direction of change. *We* must be *the* protagonists for the people's health, present and future. Remembering the inability to speak with percision and certainty about the future is no excuse for silence. While I would avoid the hyperbole of politics, and the politics of Camelot and charisma, I would ask you to accept the thought that the mobilization of feeling that is the necessary prelude to all but the most routine social action requires some stimulus stronger than sound argument.

Yes . . . if we speak honestly and effectively, we can, in Woodrow Wilson's words, "Creep into the confidence of those we would lead." A confidence not engendered in a moment but requiring tenacity, flexibility, imagination, foresight, and those other attributes defining respected judgement. I might say, parenthetically, one of those is not a sense of infallibility. Remember — "Just outside my wisdom are words that would answer everything."

As we turn increasing numbers of your medical minds to the dilemma of others, we should constantly remind each other we can not "Do it alone." We must learn to work more easily in a climate in which diverse ideas might meet. Others listen if we listen. Soliloquies create a vacuum while persuasion deals in the coin of self-interest with men and women who have some freedom to reject what they find counterfeit. This past year, we have found a real commonality of interests in working with various groups. This interest, this understanding, requires your time. Without the time of many, we (paraphrasing Thomas Hughes) will be presiding with varying effectiveness over an association of men and women, too proud to fight, too sophisticated to agree, too democratic to be directed, too assertive to acquiesce, too conservative to be led, too complicated to be consistent, and too elitist to accept the majority rule.

But I believe time and thought we will expend so that positions defined and programs designed by the Association will not be drawn with the restrictive pallet of past policies, but will instead manifest this new spirit — this new ethos. Reflecting the future — not the past; seasoned by the judgement of many ages; and, endowed with the Hippocratic idealism present in so many — expressed by so few. In matters medical, let us help the patient — the public — find a better tomorrow.

President

Maine Medical Association

1973-1974

PAUL A. FICHTNER, M.D.

Paul A. Fichtner, M.D. of Bath, Maine became the 124th President of the Maine Medical Association at the 120th Annual Session banquet on June 18, 1973. Dr. Fichtner served as M.M.A. Councilor for the Third District from 1970 to 1971 and was elected to represent that District (Lincoln-Sagadahoc, Knox) again on the Executive Committee for 1971 to 1974, serving as its Chairman for 1971 to 1972, and was elected President-elect for 1972 to 1973.

Dr. Fichtner was born in Hartford, Connecticut on December 10, 1920, son of Paul A. and Frances K. Fichtner. He was graduated from Trinity College in 1943 and received his medical degree from Long Island College of Medicine in 1946. He interned at the Hartford Hospital from 1946 to 1947 and served a general surgery residency at McCook Memorial Hospital in Hartford from 1947 to 1948. Dr. Fichtner served in the U. S. Army Medical Corps as a Captain from 1948 to 1951. He was located in Rangeley, Maine from 1951 to 1962 and then moved to Bath where he now practices.

He is a member of the Lincoln-Sagadahoc County Medical Society, the Maine Medical Association, the American Medical Association and the American Academy of Family Physicians. Dr. Fichtner has served on the M.M.A. Health Insurance Committee, has been a Delegate from Franklin and Lincoln-Sagadahoc Counties, and is a medical examiner for the State of Maine.

Dr. Fichtner is married to the former Arlene Verrill.

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Kennebunkport, Maine — June 16, 17, 18, 19, 1973

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(Jan. 1, 1974 to Jan. 1, 1976)
BRINTON T. DARLINGTON, M.D.
Augusta

Speaker of the House
GEORGE W. BOSTWICK, M.D.
Newcastle

DR. FICKER

DR. AMREIN

DR. OUELLETTE

DR. MCAFEE

DR. BOSTWICK

Dr. John B. Madigan was elected President-elect. Dr. Robert F. Ficker was reelected to the Executive Committee (First District) for a three-year term and elected to serve as Chairman for 1973-1974. Dr. H. Carl Amrein was also reelected (Fifth District) for a three-year term and Dr. William C. Bromley elected to the Executive Committee (Sixth District) for a three-year term. Dr. Benoit Ouellette of the Ninth District was elected to complete Dr. John B. Madigan's unexpired term (1973-1975). Dr. Robert E. McAfee was elected as Delegate to the AMA and Dr. Brinton T. Darlington, Alternate Delegate to the AMA. Dr. George W. Bostwick was reelected Speaker of the House of Delegates.

DR. MADIGAN of Houlton, Maine was born in Houlton on May 11, 1917, son of James Cottrill and Doris Waterall Madigan. He was graduated from Georgetown University in 1938 and received his medical degree from Tufts University School of Medicine in 1942. He served a rotating internship at the Springfield Hospital in Massachusetts from 1942 to 1943 and then served in the U. S. Army for three years as a Captain. In 1946, he served a residency at the Springfield Hospital and then located in Houlton in 1947.

He is a member of the Aroostook County Medical Society, the Maine Medical Association, and the American Medical Association. He is a former President of the Aroostook Tuberculosis Association, a member of the Maine Chapter of the American Academy of Family Physicians, a former member of the Houlton Town Council, and the N.E. Regional Medical Advisory Board, and is a Board Director of the Maine Health Planning Council, the Aroostook Health Services, Inc. and the Cary Memorial Library.

Dr. Madigan was married in 1952 to the former Mildred Moriarty of Quincy, Massachusetts and they have seven children.

DR. FICKER of Kennebunkport, Maine was born in Brooklyn, New York on April 14, 1916, son of Robert V. and Emily Kaiser Ficker. He was graduated with a B.A. degree from Bard College of Columbia University in 1939 and received his medical degree from New York University College of Medicine in 1943. He interned at Bellevue Hospital in New York from 1942 to 1943 and at the Brooklyn Hospital from 1943 to 1944. Dr. Ficker served as a Captain in the U. S. Army Medical Corps for two years. He took a postgraduate course in Internal Medicine at New York University from 1946 to 1947, and then practiced in Wanaque and Ogdensburg, New Jersey before coming to Kennebunkport in 1953. He has been on the staff of Webber Hospital in Biddeford since 1953.

He is a member of the York County Medical Society, serving as President in 1960, the Maine Medical Association and the American Medical Association. Dr. Ficker is a current member of the M.M.A. Peer Review Committee and was Chairman of the School Health Committee from 1958 to 1959. In addition, Dr. Ficker has been a member of the M.M.A. House of Delegates for several years.

DR. BROMLEY of Ellsworth, Maine was born in Jackson, Michigan on June 7, 1934, son of John P. and Augusta W. Bromley. He was graduated from Michigan State University in 1955, received his medical degree from the University of Michigan Medical School in 1959, and served a residency in Ophthalmology there from 1969 to 1971. He served in the U. S. Air Force as a Captain from 1960 to 1963, practiced in Southwest Harbor for 5 years, and then located in Ellsworth.

He is a member of the Hancock County Medical Society, the Maine Medical Association and is a member of the courtesy staff of the Maine Coast Memorial Hospital, the Mt. Desert Island Hospital and the Blue Hill Memorial Hospital. Dr. Bromley was certified by the American Board of Ophthalmology in 1973.

Dr. Bromley is married to the former Julie Wass. They have two sons, Thomas and Steven, and two daughters, Jane and Mary Ellen.

DR. OUELLETTE of Fort Kent, Maine was born in Baker Lake, New Brunswick, Canada on June 8, 1925. He was graduated from St. Joseph University in Moncton, New Brunswick and received his medical degree in 1954 from Laval University Faculty of Medicine in Quebec. He served in the Canadian Army Medical Corps in Canada and Europe as a Captain from 1954 to 1959, and then located in Fort Kent where he now practices.

He is a member and Secretary of the Aroostook County Medical Society, a member of the Maine Medical Association, the American Medical Association and the New Brunswick Medical Society.

Dr. Ouellette is married to the former Blanche Couillard and they have two children, ages 19 and 13.

DR. McAFEE of Portland, Maine was born in Portland on August 25, 1935, son of Harold G. and Elizabeth H. McAfee. Dr. McAfee was graduated from Deering High School in 1952, Bates College in 1956 and received his medical degree from Tufts University School of Medicine in 1960. He interned at the Maine Medical Center and served a residency in General Surgery from 1961 to 1965. Since 1965, Dr. McAfee has been attending surgeon at the Maine Medical Center and the Mercy Hospital, and consultant in surgery at the Pineland Hospital and Training Center since 1971.

He is a member of the Cumberland County Medical Society, the Maine Medical Association, the American Medical Association, and is certified by the American Board of General Surgery.

Dr. McAfee is currently President-elect of the American Cancer Society, Maine Division, Inc. and is also serving as Chairman of its Medical Affairs Committee; Chairman of the State Advisory Committee and State Ambulance Licensure Board of the Emergency Medical Services Division, Department of Health and Welfare; a member of the Committee on Recruitment, Aid and Placement of the Maine Medical Association, serving as Chairman (1971-1972); and is clinical instructor in surgery at Tufts University School of Medicine.

Active in community affairs, he is Chairman of the Sex Education Advisory Committee, City of Portland School Committee, and President of the Forest Lake Improvement Association.

DR. DARLINGTON of Augusta, Maine was born in Olean, New York on January 7, 1922, son of J. Leon and Martha T. Darlington. He was graduated from Ohio University in 1943 and received his medical degree from State University of New York Upstate Medical Center, Syracuse in 1945. He interned at St. Luke's Hospital in Cleveland, Ohio and served a residency at the U. S. Marine Hospital in Staten Island. In 1952, he located in Augusta.

He is a member of the Kennebec County Medical Association, the Maine Medical Association and the American Medical Association. Dr. Darlington was a former director of the National Tuberculosis Association, former President of the Maine Thoracic Society, the Maine Tuberculosis and Health Association and the Maine Society of Internal Medicine.

State Of Maine

Department of Health and Welfare

DEAN H. FISHER, M.D.
COMMISSIONER

Re-Evaluation of Current Tuberculosis Control Practices

JERRY M. HERSHOVITZ*

Since the turn of the century, great strides have been made in the control of tuberculosis. In 1972, there were only 87 newly reported active tuberculosis cases in Maine, with an active case rate of 8.8/100,000 which ranks among the lowest in the nation (37th position). Our statistics are impressive and tend to lend credence to the popular belief that, in our State, tuberculosis is near eradication. Nevertheless, there are those among us that still consider tuberculosis to be a problem, for in the past 10 years our newly reported case rate has remained relatively constant. Perhaps this is because we have failed to recognize certain epidemiologic facts that dictate a change in our current tuberculosis control practices so that some day we may speak of eradication rather than of control. The time has come for us to re-evaluate these practices and refine them if we are to make more effective use of our present knowledge.

In calendar year 1972, 868 clinically inactive tuberculosis patients were scheduled for annual x-rays and examinations in compliance with State policy which requires the routine follow-up of inactive patients for ten years. The purpose of long-term follow-up is to identify patients with reactivated tuberculosis before they develop serious complications and infect others. Yet, records in the Division of Tuberculosis Control present evidence that belies the need for long-term follow-up.

In 1972, 6 patients in Maine developed recurrent disease, and by current standards none had received adequate chemotherapy, nor had they been discovered by routine examination. An analysis of additional data compiled by the Bangor-Brewer Tuberculosis and Health Association disclosed that only 3 reactivations were identified since 1968 through routine annual x-rays and examinations of the inactive patients seen at their clinic. Identifying these 3 reactivations involved the examination of over 1000 inactive patients yearly.

*Administrative Director, Division of Tuberculosis Control, Maine Department of Health and Welfare.

It has also been our experience that recurrent disease is rarely found at the time of annual examination. Almost always, it is identified when a patient becomes sick and seeks medical attention on his own. There are cases on record of patients that have become sick but delayed seeking treatment because their annual examination was only 2 or 3 months away.

Therefore, the time has come to critically evaluate the practice of routine follow-up of inactive tuberculosis patients that have had adequate chemotherapy. Edsall and Collins[1] reporting of the routine follow-up of inactive patients state that the reactivation rate after adequate chemotherapy is less than one percent. When our judgment leads us to believe that a patient has taken his medicine as prescribed and he has progressed satisfactorily, should we not dismiss him from follow-up and ask only that he returns if he becomes sick? Patients that have not completed chemotherapy as prescribed should, of course, remain under surveillance, but rather than continue to examine these people year after year, why not treat them?

To continue the practice of long-term follow-up of inactive patients is both unproductive and costly.[2] We have learned from experience that it is far better to concentrate our efforts where the cost/effectiveness ratio is in far better balance. For example, how many people must we examine to find one active case of tuberculosis?

In one large city elsewhere, every 55 household contacts examined produced one active case. Among non-household contacts, 416 had to be examined for the same yield. In Maine, in 1972, the examination of 169 household contacts uncovered 2 active cases; the examination of 529 non-household contacts did not uncover one case. The tuberculin reactor rate among household contacts was 30.2%, but among our non-household contacts it was only 10.3%. The tuberculin reactor rate among household contacts exposed to active patients with positive smears was 41%.

The risk of household contacts, both tuberculin negative (especially children) and tuberculin

positive, developing active disease during the first year after discovery of the index case is about 1 in 30. Household contacts are at the highest risk of developing active tuberculosis. In 1972, the national yield of active cases from examination of household contacts was 2030/100,000 compared with an overall national case rate of 15.8/100,000. For household contacts under 15 years of age, the rate was 3450/100,000.[3] These statistics emphasize the need and importance of prompt examination of household contacts. It also presents a strong argument for discontinuing the epidemiologic follow-up of non-household contacts, except in special circumstances where exposure has been close and prolonged.

Also worthy of our attention is the practice of tuberculin testing school personnel in our public schools. This group is important because of the consequence of their being infectious in a closed environment with children. We spend considerable time and effort through skin testing programs in order to identify those infected, but all too often physicians do not place their tuberculin reactors on preventive treatment. Recently, there was the experience of a teacher in Maine infecting 5 other teachers and 19 students. Six of the students developed active disease before they were identified. Fortunately, however, everyone involved did receive chemotherapy or chemoprophylaxis (isoniazid). The teacher had been a known tuberculin reactor for several years. I suggest that teachers and other school personnel identified as tuberculin reactors and in close contact with children be seriously considered for isoniazid if we hope to prevent such occurrences in the future.

We cannot expect to place every contact and reactor on chemoprophylaxis, however, priorities must be set for the protection of the public's health. In establishing these priorities, consideration must be given to the relative risk of developing active disease, and the opportunities for infecting others. These must be weighed against the relative risk of preventive treatment.

In summary, I have presented the following recommendations to the Board of Tuberculosis Consultants, Division of Tuberculosis Control, for their review and consideration:

1. Discontinue the long-term follow-up of adequately treated tuberculosis patients.
2. Place priority on the early diagnosis of tuberculosis suspects and on the immediate examination of household contacts by establishing time perimeters.
3. Place all school personnel that are tuberculin reactors and in contact with children on isoniazid for one year, except when contraindicated for health reasons.

REFERENCES

1. Edsall, J. and Collins, G.: Routine Follow-up if Inactive Tuberculosis, A Practice to be Abandoned, Amer. Rev. Resp. Dis., 1973, 107, 851.
2. Bailey, W. C., Thompson, Doris H., Jacobs, S., Ziskind, M., and Greenberg, H. B.: Evaluating the Need for Periodic Recall and Re-examination of Patients with Inactive Pulmonary Tuberculosis, Amer. Rev. Resp. Dis., 1973, 107, 855.
3. Center for Disease Control: Tuberculosis Program Reports.

HYPERTROPHIC PYLORIC STENOSIS IN THE ADULT – *Continued from Page 175*

physiologic and fewer problems associated with the dumping syndrome. The Fredet-Ramstedt procedure, which is used in infants, is not generally accepted as the possibility of diverticulum formation is present. Other forms of therapy have been pyloric dilatation vagotomy and pyloroplasty, and gastroenterostomy.

REFERENCES

1. Belding, H.H., Kernohan, J.H.; A Morphologic Study of the Myenteric 'Plexus and Musculature of the Pylorus with Special References to Changes in Hypertrophic Pyloric Stenosis. Surg. Gynec. and Obst. 97: 322-354, 1953.
2. Christiansen, K., Grantham, A., Idiopathic Hypertrophic Pyloric Stenosis in the Adult. Arch. Surg. 85: 207-213, 1962.
3. Craver, W.L., Hypertrophic Pyloric Stenosis in Adults. Gastroenterology. 33: 914-924, 1957.
4. Hakhamimi, H., Cogbill, C., Primary Adult Hypertrophic Pyloric Stenosis. Case Report and Review of Literature. Am. Surgeon. 34: 446-449, 1968.
5. Hiebert, B.W., Farris, J.M., Hypertrophic Pyloric Stenosis in the Adult. Review of 22 Cases. Am. Surgeon. 32: 712-714, 1966.
6. Kirklin, B.R., Harris, M.T., Hypertrophy of the Pyloric Muscle of Adults: A Distinctive Roentgenologic Sign. Am. J. Roentgenol. 29: 437-442, 1933.
7. Knight, C.D., Hypertrophic Pyloric Stenosis in the Adult. Am. Surg. 153: 899-910, 1961.
8. Mack, H.C., Adult Hypertrophic Pyloric Stenosis. Report of 2 Cases with Variable X-ray Findings. Arch. Int. Med. 104: 574-579, 1959.
9. McCann, J.C., Dean, M.A., Hypertrophy of the Pyloric Muscle in Adults. Experiences with Conservative and Radical Surgical Treatment. Surg. Gynec. and Obst. 90: 534-542, 1950.
10. Seaman, W.B., Focal Hypertrophy of the Pyloric Muscle. Torus Hyperplasia. Am. J. Roentgen. 96: 388-392, 1966.
11. Seaman, W.B., Hypertrophy of the Pyloric Muscle In Adults. Radiology, 80: 753-764, 1963.
12. Skoryna, S., Dolan, H. Gley, A., Development of Primary I Pyloric Hypertrophy in Adults in Relation to the Structure and Function of the Pyloric Canal. Surg, Gynec, and Obst. 108: 83-92, 1959.

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ROCHE announces new BACTRIM™

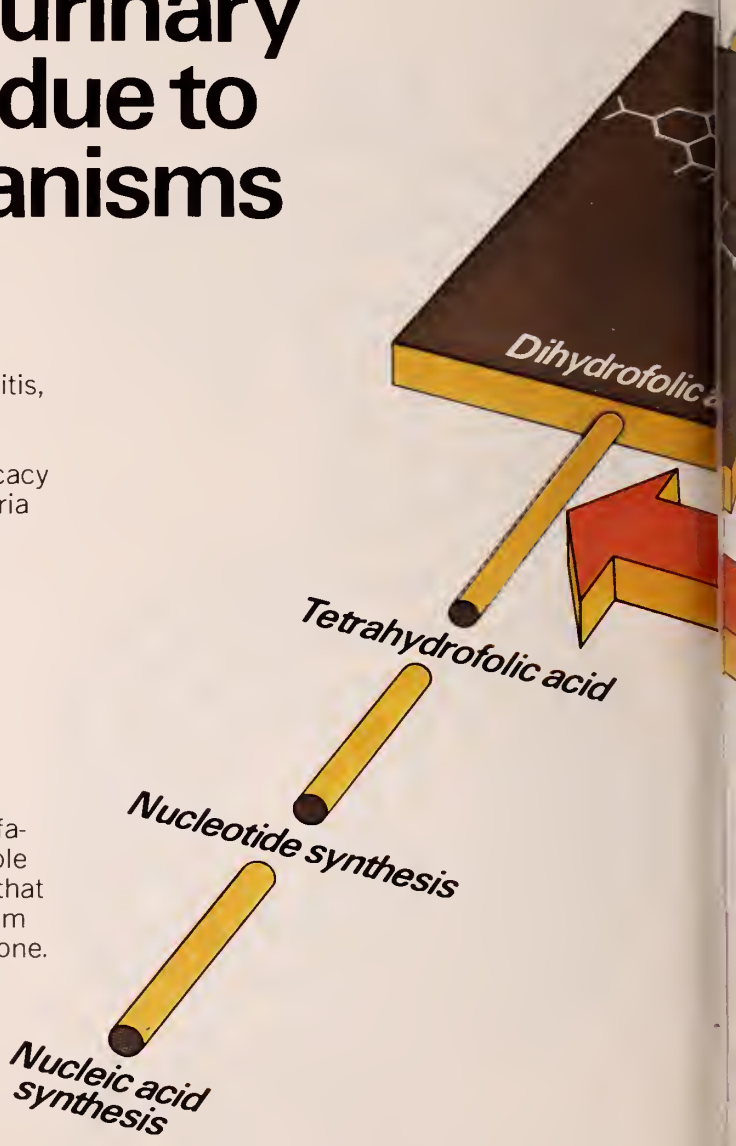
Each tablet contains 80 mg trimethoprim and 400 mg sulfamethoxazole.

a new type of antibacterial for a two-pronged attack against chronic urinary tract infections due to susceptible organisms

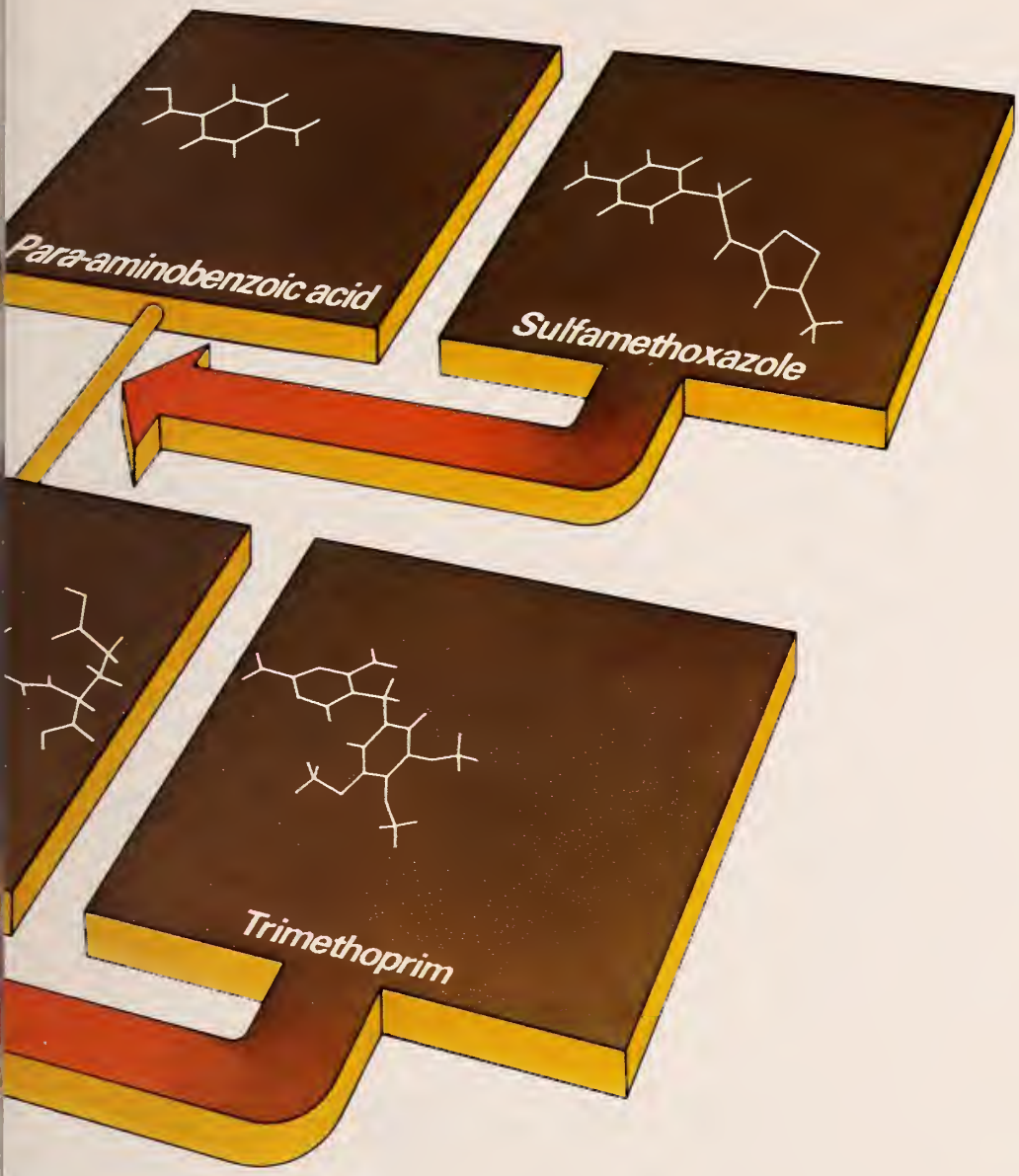
Bactrim is highly effective in the treatment of these infections—primarily pyelonephritis, pyelitis and cystitis, when due to susceptible organisms (usually *E. coli, Klebsiella-Enterobacter, Proteus mirabilis,* and, less frequently, indole-positive proteus species). This efficacy is related to the unique mode of action against bacteria (see opposite page), an action that, in effect, makes Bactrim a new type of antibacterial.

Bactrim significantly superior to constituents in patients with obstructive complications

In the presence of obstructive uropathy, Bactrim has demonstrated efficacy which is superior to either sulfamethoxazole or trimethoprim alone against susceptible organisms. In addition, *in vitro** studies have shown that bacterial resistance develops more slowly with Bactrim than with either trimethoprim or sulfamethoxazole alone.



*Please note that clinical conclusions cannot be extrapolated from *in vitro* studies.



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interrupts life cycle of susceptible bacteria

Unique mode of action interrupts the life cycle at two important points, thereby impeding the production of nucleic acids and proteins essential to these bacteria. These consecutive interruptions occur because sulfamethoxazole and trimethoprim resemble naturally existing substrates. By competitive replacement of these substrates, they inhibit further synthesis.

new **BACTRIM**™

Each tablet contains 80 mg trimethoprim and 400 mg sulfamethoxazole.

for chronic urinary tract infections

Before prescribing, please see complete product information on last page of advertisement.

Excellent clinical response in chronic urinary tract infections

A multiclinic, double-blind study* of response to a ten-day course of therapy in 471† patients with chronic urinary tract infections demonstrated the superiority of Bactrim. On the 10th day after initiation of therapy, 91.7% (of 168 patients) showed significant bacteriological response to Bactrim compared with 81.2% (of 144 patients) to trimethoprim and 64.5% (of 155 patients) to sulfamethoxazole. In patients with obstructive complications, 10th day response was 94.8% (of 97 patients) to Bactrim, 72.9% (of 85 patients) to trimethoprim and 58.5% (of 94 patients) to sulfamethoxazole.

Excellent response maintained

Bactrim proved equally impressive in maintaining this bacteriological response. In the above study, after ten-day therapy with Bactrim, 68.4% of patients with chronic urinary tract infections maintained response for up to 42 consecutive days, compared with 59.7% with trimethoprim and 44.4% with sulfamethoxazole. In patients with obstruction, 70.8% of those on Bactrim maintained response for up to 42 consecutive days, compared with 49.4% on trimethoprim and 38.8% on sulfamethoxazole. The figures are particularly remarkable in cases with urinary obstruction – cases regarded as being notoriously difficult to treat.

To date, low incidence of significant side effects

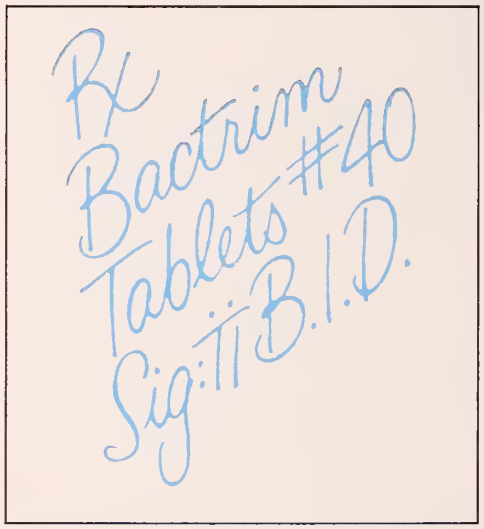
Although Bactrim demonstrated impressive clinical results, it is important to note that the incidence of clinically significant adverse effects was low, mainly nausea and/or vomiting, rash, leukopenia, SGOT increase and creatinine increase.

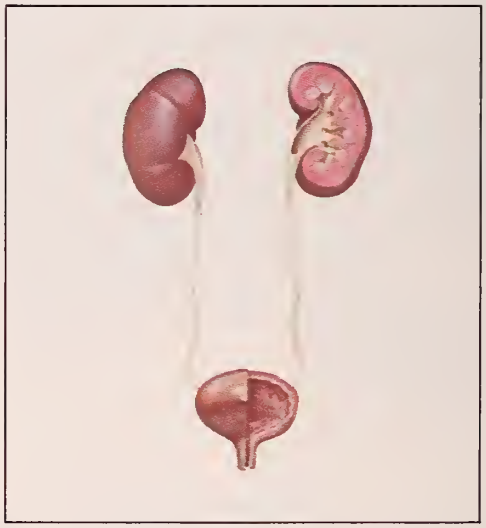
Bactrim should be given with caution to patients with impaired renal or hepatic function, possible folate deficiency and to those with severe allergy or bronchial asthma. Adequate fluid intake must be maintained. Complete blood counts, urinalyses with careful microscopic examination, and renal function tests should be performed during therapy.

Currently, the increasing frequency of resistant organisms is a limitation of the usefulness of all antibacterial agents, especially in the treatment of chronic and recurrent urinary tract infections.

Usual adult dosage: two tablets every twelve hours for 10 to 14 days; no loading dose required.

* Data on file, Hoffmann-La Roche Inc., Nutley, N.J. 07110
† 4 patients not available for evaluation at day 10.





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for chronic urinary tract infections

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Before prescribing, please consult complete product information on facing page.

Complete Product Information:

Description: Bactrim is a synthetic antibacterial combination product, available in scored light-green tablets, each containing 80 mg trimethoprim and 400 mg sulfamethoxazole.

Trimethoprim is 2,4-diamino-5-(3,4,5-trimethoxybenzyl) pyrimidine. It is a white to light-yellow, odorless, bitter compound with a molecular weight of 290.3.

Sulfamethoxazole is N^1-(5-methyl-3-isoxazolyl) sulfanilamide. It is an almost white in color, odorless, tasteless compound with a molecular weight of 253.28.

Actions: *Microbiology:* Sulfamethoxazole inhibits bacterial synthesis of dihydrofolic acid by competing with *para*-aminobenzoic acid. Trimethoprim blocks the production of tetrahydrofolic acid from dihydrofolic acid by binding to and reversibly inhibiting the required enzyme, dihydrofolate reductase. Thus, Bactrim blocks two consecutive steps in the biosynthesis of nucleic acids and proteins essential to many bacteria.

In vitro studies have shown that bacterial resistance develops more slowly with Bactrim than with trimethoprim or sulfamethoxazole alone.

In vitro serial dilution tests have shown that the spectrum of antibacterial activity of Bactrim includes the common urinary tract pathogens with the exception of *Pseudomonas aeruginosa*. The following organisms are usually susceptible: *Escherichia coli, Klebsiella-Enterobacter, Proteus mirabilis* and indole-positive proteus species.

Representative Minimum Inhibitory Concentration Values for Bactrim-Susceptible Organisms
(MIC—mcg/ml)

Bacteria	Trimethoprim alone	Sulfamethoxazole alone	TMP/SMX (1:20) TMP	SMX
Escherichia coli	0.05—1.5	1.0 —245	0.05—0.5	0.95— 9.5
Proteus spp. indole positive	0.5 —5.0	7.35 —300	0.05—1.5	0.95—28.5
Proteus mirabilis	0.5 —1.5	7.35 — 30	0.05—0.15	0.95— 2.85
Klebsiella-Enterobacter	0.15—5.0	0.735—245	0.05—1.5	0.95—28.5

Human Pharmacology: Bactrim is rapidly absorbed following oral administration. The blood levels of trimethoprim and sulfamethoxazole are similar to those achieved when each component is given alone. Peak blood levels for the individual components occur one to four hours after oral administration. The half-lives of sulfamethoxazole and trimethoprim, 10 and 16 hours respectively, are relatively the same regardless of whether these compounds are administered as individual components or as Bactrim. Detectable amounts of trimethoprim and sulfamethoxazole are present in the blood 24 hours after drug administration. Free sulfamethoxazole and trimethoprim blood levels are proportionately dose-dependent. On repeated administration, the steady-state ratio of trimethoprim to sulfamethoxazole levels in the blood is about 1:20.

Sulfamethoxazole exists in the blood as free, conjugated and protein-bound forms; trimethoprim is present as free, protein-bound and metabolized forms. The free forms are considered to be the therapeutically active forms. Approximately 44 percent of trimethoprim and 70 percent of sulfamethoxazole are protein-bound in the blood. The presence of 10 mg percent sulfamethoxazole in plasma decreases the protein binding of trimethoprim to an insignificant degree; trimethoprim does not influence the protein binding of sulfamethoxazole.

Excretion of Bactrim is chiefly by the kidneys through both glomerular filtration and tubular secretion. Urine concentrations of both sulfamethoxazole and trimethoprim are considerably higher than are the concentrations in the blood. When administered together as in Bactrim, neither sulfamethoxazole nor trimethoprim affects the urinary excretion pattern of the other.

Indications: Chronic urinary tract infections (primarily pyelonephritis, pyelitis and cystitis) due to susceptible organisms (usually *E. coli, Klebsiella-Enterobacter, Proteus mirabilis*, and, less frequently, indole-positive proteus species).

***Important note:* Currently, the increasing frequency of resistant organisms is a limitation of the usefulness of all antibacterial agents, especially in the treatment of chronic and recurrent urinary tract infections.**

Contraindications: Hypersensitivity to trimethoprim or sulfonamides. Pregnancy and during the nursing period (see Reproduction Studies).

Warnings: Deaths associated with the administration of sulfonamides have been reported from hypersensitivity reactions, agranulocytosis, aplastic anemia and other blood dyscrasias. Experience with trimethoprim alone is much more limited, but it has been reported to interfere with hematopoiesis in occasional patients. In elderly patients concurrently receiving certain diuretics, primarily thiazides, an increased incidence of thrombopenia with purpura has been reported.

The presence of clinical signs such as sore throat, fever, pallor, purpura or jaundice may be early indications of serious blood disorders. Complete blood counts should be done frequently in patients receiving Bactrim. If a significant reduction in the count of any formed blood element is noted, Bactrim should be discontinued.

At the present time, there is insufficient clinical information on the use of Bactrim in infants and children under 12 years of age to recommend its use.

Precautions: Bactrim should be given with caution to patients with impaired renal or hepatic function, to those with possible folate deficiency and to those with severe allergy or bronchial asthma. In glucose-6-phosphate dehydrogenase-deficient individuals, hemolysis may occur. This reaction is frequently dose-related. Adequate fluid intake must be maintained in order to prevent crystalluria and stone formation. Urinalyses with careful microscopic examination and renal function tests should be performed during therapy, particularly for those patients with impaired renal function.

Adverse Reactions: For completeness, all major reactions to sulfonamides and to trimethoprim are included below, even though they may not have been reported with Bactrim.

Blood dyscrasias: Agranulocytosis, aplastic anemia, megaloblastic anemia, thrombopenia, leukopenia, hemolytic anemia, purpura, hypoprothrombinemia and methemoglobinemia.

Allergic reactions: Erythema multiforme, Stevens-Johnson syndrome, generalized skin eruptions, epidermal necrolysis, urticaria, serum sickness, pruritus, exfoliative dermatitis, anaphylactoid reactions, periorbital edema, conjunctival and scleral injection, photosensitization, arthralgia and allergic myocarditis.

Gastrointestinal reactions: Glossitis, stomatitis, nausea, emesis, abdominal pains, hepatitis, diarrhea and pancreatitis.

C.N.S. reactions: Headache, peripheral neuritis, mental depression, convulsions, ataxia, hallucinations, tinnitus, vertigo, insomnia, apathy, fatigue, muscle weakness and nervousness.

Miscellaneous reactions: Drug fever, chills, and toxic nephrosis with oliguria and anuria. Periarteritis nodosa and L. E. phenomenon have occurred.

The sulfonamides bear certain chemical similarities to some goitrogens, diuretics (acetazolamide and the thiazides) and oral hypoglycemic agents. Goiter production, diuresis and hypoglycemia have occurred rarely in patients receiving sulfonamides. Cross-sensitivity may exist with these agents. Rats appear to be especially susceptible to the goitrogenic effects of sulfonamides, and long-term administration has produced thyroid malignancies in the species.

Dosage and Administration: Not recommended for use in children under 12 years of age.

The usual adult dosage is two tablets every 12 hours for 10 to 14 days.

For patients with renal impairment:

Creatinine Clearance (ml/min)	Recommended Dosage Regimen
Above 30	Usual standard regimen
15-30	2 tablets every 24 hours
Below 15	Use not recommended

How Supplied: Tablets, containing 80 mg trimethoprim and 400 mg sulfamethoxazole—bottles of 100 and 500; Tel-E-Dose® packages of 1000; Prescription Paks of 40, available singly and in trays of 10. Imprint on tablets: ROCHE 50.

Reproduction Studies: In rats, doses of 533 mg/kg sulfamethoxazole or 200 mg/kg trimethoprim produced teratological effects manifested mainly as cleft palates. The highest dose which did not cause cleft palates in rats was 512 mg/kg sulfamethoxazole or 192 mg/kg trimethoprim when administered separately. In two studies in rats, no teratology was observed when 512 mg/kg of sulfamethoxazole was used in combination with 128 mg/kg of trimethoprim. However, in one study, cleft palates were observed in one litter out of 9 when 355 mg/kg of sulfamethoxazole was used in combination with 88 mg/kg of trimethoprim.

In rabbits, trimethoprim administered by intubation from days 8 to 16 of pregnancy at dosages up to 500 mg/kg resulted in higher incidences of dead and resorbed fetuses, particularly at 500 mg/kg. However, there were no significant drug-related teratological effects.

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—News From Blue Cross and Blue Shield—

Why Regional Blood Banks?

Regional Blood Banks, Inc. is the only centralized blood banking system in the State of Maine. A non-profit organization, Regional Blood Banks, Inc. operates on the same concept as the Red Cross Blood Banks in other states. The program is composed of a completely voluntary donor system and centralized Blood Bank facilities and will be able to meet all the blood needs of the entire State in the future. Begun in Waterville four years ago, Regional has grown to serve 26 hospitals in the Portland, Auburn, and Waterville areas and will soon be serving in the Bangor area.

By establishing area blood banks capable of supplying all the member hospitals in their areas with their daily blood needs, the Regional Blood Bank system has improved on the old system whereby each hospital had to find and maintain its own blood resources. Now, through area blood banks, the majority of the hospitals in the State will no longer have to concern themselves with the problems of finding donors and drawing blood for their patients.

Historically, hospitals have had difficulty in encouraging the public to give blood and have had to provide such incentives as penalty charges to replace blood. Under the typical penalty charge system, a patient had the choice of paying a fee for the blood or having friends and relatives replace the blood used, usually at the rate of two pints for each one used. Some hospitals also had to resort to buying out-of-state commercial blood at one time or another to provide the full needs of Maine patients. Commercial blood has been known to be of less than the highest quality.

With the advent of the Regional concept, the need for individual hospitals to purchase and maintain blood processing equipment and employ staff to operate the equipment was overcome. Regional Blood Banks are staffed with trained personnel who screen potential donors to ensure that only healthy blood is taken. They are experienced in testing the blood for diseases which could affect the recipient. The blood, once typed and tested, is rotated within the State system to effect the most efficient use of the precious commodity.

An increasing number of hospitals are participating in blood component therapy, which consists of treatment of certain blood deficiencies and diseases with only those parts of the blood, such as red blood cells or plasma, necessary to the prescribed treatment. Regional, with its ability to break blood down into its components, allows all member hospitals the opportunity to provide patients with component therapy. This capability, once only available in the larger hospitals, is now available on request in more remote communities, saving many patients the expense of having to travel to a larger hospital and, in many cases, allowing them to be treated on an outpatient basis in their own communities instead of on an inpatient basis away from home.

Regional Blood Banks, Inc., as a non-profit organization, supports itself on a service charge which covers operational expenses such as the drawing and processing expenses. This service charge has always been passed on to the patient by the hospital as a service charge for laboratory work and usually is covered through the patients health coverage program such as Blue Cross and Blue Shield. The Regional service charge, then, is essentially the hospital laboratory changes for blood drawing and processing taken out of the hospital context and then returned to the hospital bill by Regional. The advantages of having a centralized drawing and processing center are passed on to all patients in Maine.

In May 1973, an administrative agreement was reached between Maine Blue Cross and Blue Shield and Regional Blood Banks, Inc. which allows Maine Blue Cross and Blue Shield to give administrative support to the Regional organization. At such time as Regional is able to handle all of its administrative requirements, it will be able to again operate completely on its own. In explanation of the Blue Cross support, Richard F. Nellson, the President of the organization, stated at the contract signing; "We have supported the Regional Blood Bank concept since its inception over four years ago, and we consider the program a necessity for the State of Maine. We will do everything in our power to stimulate the growth in membership of Regional Blood Banks, and we encourage every healthy Maine citizen to give at least one pint of blood a year to insure that no one will ever lose his life for want of blood in Maine."

Mr. Nellson went on to say that: "Blue Cross and Blue Shield members who give blood through the Regional program will incur absolutely no expenses should they require blood. The blood is free, and the service charge is absorbed by their Blue Cross coverage."

The Regional Blood Bank program, as a part of Statewide drive to coordinate health care facilities, has received the endorsement of the Maine Hospital Association, Maine Comprehensive Health Planning Agency, Maine Medical Asso-

ciation, Maine Osteopathic Association, all the contracting hospitals and the many pathologists who have been active in the program, and is a certified member of the American Association of Blood Banks. Its goal of providing good, free blood to all the hospitals in Maine is not too far from being realized. With your help, this completely voluntary donor program and blood banking system will be able to continue to fill a basic need which can only be met with personal commitments of time and blood.

THE DIAGNOSIS AND MANAGEMENT OF THE ANEMIA ASSOCIATED WITH CARDIAC PROSTHETIC VALVES — *Continued from Page 178*

6. Sears, D. A., Crosby, W. H.: Intravascular hemolysis due to intracardiac prosthetic devices. Diurnal variations related to activity. Amer. J. Med. 39: 341, 1965.
7. Eyster, E.: Traumatic hemolysis with hemoglobinuria due to ball valve variance. Blood. 33: 391, 1969.
8. Myhre, E., Dale, J., Rasmussen, K.: Erythrocyte destruction in different types of Starr-Edwards aortic ball valves. Circulation 42: 515, 1970.
9. Santinga, J. T., Kirsh, M. M.: Hemolytic anemia in series 2300 and 2310 Starr-Edwards prosthetic valves. Ann. of Thor. Surg. 14: 539, 1972.
10. Eyster, E., Rothchild, J., Mychajliw, O.: Chronic intravascular hemolysis after aortic valve replacement. Circulation 44: 657, 1971.
11. Grosse-Brockoff, F., Gehrmann, G.: Mechanical hemolysis in patients with valvular heart disease and valve prostheses. Am. Heart J. 74: 137, 1967.
12. Walsh, J. R., Brodeur, M. T., Ritzmann, L. W., Sutherland, D. W., Starr, A.: Urinary iron excretion in patients with prosthetic heart valves. J.A.M.A. 198: 91, 1966.
13. Eyster, E., Edgington, T. S., Liem, H. H., Muller-Eberhard, U.: Plasma hemopexin levels following aortic valve replacement: A valuable screening test for assessing the severity of cardiac hemolysis. J. Lab. Clin. Med. 80: 112, July, 1972.
14. Myhre, E., Dabe, J., and Rasmussen, K.: Quantitative aspects of hemolysis in aortic valvular disease and ball valve prostheses. Acta Med. Scand. 189: 101, 1971.
15. Bothwell, T. H., Pirzio-Birol, G., Finch, C. A.: Iron absorption. J. Lab. Clin. Med. 51, 24, 1958.
16. Crosby, W. H.: Iron metabolism. Dis. of the Month.
17. Sears, D. A., Anderson, P. R., Fay, A. L., Williams, H. L., Crosby, W. H.: Urinary iron excretion and renal metabolism of hemoglobin in hemolytic diseases. Blood 28: 708, 1966.

BLUE CROSS AND OTHER INSURANCES ARE ACCEPTED

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The Department of Postgraduate Medicine of Albany Medical College announces Reservations Now Being Accepted for the Fifteenth Postgraduate Medical Seminar Cruise January 26 — February 9, 1974

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For information write to: Frank M. Woolsey Jr., M.D., Department of Postgraduate Medicine, Albany Medical College, Albany, New York 12208.

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Dr. Robertson Elected President-Elect of the Massachusetts Society of Internal Medicine

Dr. George J. Robertson of North Andover, Massachusetts has been elected as president-elect (1973-1974) of the Massachusetts Society of Internal Medicine, an organization of 500 physicians in the Commonwealth engaged in the practice specialty of internal medicine.

The new president-elect is on the faculty of Tufts University School of Medicine and is a senior staff member at the New England Medical Center Hospital, the major clinical unit of Tufts-New England Medical Center (T-NEMC). At Tufts, Dr. Robertson is assistant professor of medicine and associate dean for continuing education.

He also serves as medical director of the Bingham Associates Fund (BAF). The BAF, founded more than a quarter-century ago to improve health in rural areas, has concentrated its efforts in the State of Maine. With Dr. Robertson's assistance, and the cooperation of others at T-NEMC, the BAF now has links with 54 Maine hospitals.

The Bingham Associates Fund traditionally has directed its limited resources toward improving health care services via the development of regional relationships between T-NEMC and regional hospital centers and community hospitals in Maine.

The North Andover physician also is project director of a multi-million dollar Area Health Education Center (AHEC) sponsored by the Bureau of Health Manpower Education of the National Institutes of Health. This program, which links Tufts Medical School and Maine Medical Center in Portland, has the mandate to develop better distribution of health manpower through educational programs for medical students at the AHEC centers which are at a major distance from medical schools. The AHEC program is the largest in Tufts history to train health manpower, and the only one of 11 national AHEC centers based in a private medical school.

Dr. Robertson is a graduate of both Tufts University and its Medical School. After three years of graduate training in internal medicine at Boston City Hospital and New England Medical Center Hospital, he went to Waterville, Maine, and practiced full time for 15 years.

While in Maine, he served as medical director of the Sisters Hospital and director of medical education at the Thayer Hospital, both in Waterville. In 1962, he received the Maine Medical Association's award for outstanding service to the community.

In 1963, he returned to Massachusetts and to the faculty and staff of the Tufts University School of Medicine and New England Medical Center Hospital, after being named medical director of the Bingham Associates Fund.

Secretary's Report
Annual Meeting of Maine Neurosurgical Society
June 18, 1973

The annual meeting of the Maine Neurosurgical Society was held in conjunction with the annual meeting of the Maine Medical Association in Kennebunkport, Maine, on June 18, 1973. Present were Daniel A. Rock, M.D., President; Jose Rodriguez, M.D.; Julius Ciembroniewicz, M.D. and Richard M. Swengel, M.D., Secretary-Treasurer. During the discussion of old business, it was further reported on the onsite peer review team to Thayer Hospital in Waterville during the month of April 1973. Plans were also finalized for the onsite peer review team's meeting at Augusta General Hospital on September 5. The team is to meet at Augusta General Hospital in the private dining room at about 5:00 p.m. of that date.

There were several items of business discussed during new business. The first was a report by Doctor Rock of the annual June meeting of the New England Neurosurgical Society in Boston. He presented discussion of the data presented by Dr. Ernest Sacks of Hanover in relation to regional PSRO for neurological surgery as proposed by the American Association of Neurological Surgeons and Congress of Neurological Surgeons. Apparently, the plan for the New England area is to regionalize Connecticut and Rhode Island, to have Massachusetts stand alone, and to have the northern tier states of Vermont, New Hampshire, and Maine embodied into a third region.

Letter to Doctor Fichtner, President of the Maine Medical Association, from Wallace E. Brown, Deputy Secretary of State, Motor Vehicle Division, was forwarded to the Society for action. This was in the form of a request to provide guidelines to the Motor Vehicle Division concerning length of time necessary in a seizure-free state to be eligible to apply for or hold a valid driver's license. Doctor Rock has agreed to contact various national agencies and the medical neurologists in the State of Maine in an attempt to provide some firm guideline.

Also under new business, there was discussion concerning use of the Congress of Neurological Surgeons' Utilization Review Guideline published in 1969 as minimum standards for the peer review process as established within this Society.

The Treasurer's Report for the Maine Neurosurgical Society from June 1972 through June 1973 was presented, discussed, and accepted.

Scientific session was a discussion of the indications for angiography in patients with cerebral vascular disease. Doctor Rock presented a discussion centered around the guidelines presented by the Joint Committee for Stroke Facilities published in the journal of *STROKE*, Volume 4, January-February, 1973. A lively discussion ensued with the members of the committee present who felt the guidelines were quite excellent, and these were being followed in general in the centers represented.

RICHARD M. SWENGEL, M.D.
Secretary-Treasurer

Letter to the Editor

To the Editor:

Survey of Neonatal Mortality in Maine — 1967-1971

In the near future, hospitals in the State of Maine will receive requests for clinical data on more than two hundred and fifty newborn infants who died in Maine during the five-year period from 1967 through 1971. This will be the first step in a Statewide survey of neonatal mortality during this period of time.

This survey is being undertaken in an effort to improve medical services available to the newborn infant in Maine. The purpose is to identify those aspects of neonatal health care which are presently less than optimal, and to develop means of improvement. The investigation involves the joint efforts of the Division of Child Health of the State Department of Health and Welfare, and the Maternal and Child Welfare Committee of the Maine Medical Association.

The first portion of the study is directed to the area of congenital anomalies. The death certificates of all liveborn infants who died within the first month of life during the five-year period mentioned were reviewed. Of these, 225 infants, or 16%, died of congenital structural anomalies.

With this data known, we must now turn to hospital records of these children for more detailed information. Specific data will be requested from the various hospitals, and upon completion of the survey, the information obtained will be made available to all health care institutions and personnel in Maine. Specific patient data will, of course, be held in strict confidence.

We solicit your support and cooperation in this effort to improve health care services for the newborn in this State.

MARTIN J. BELL, M.D.
Surgery of Infants and Children
7 Bramhall Street
Portland, Maine 04102

County Society Notes

LINCOLN-SAGADAHOC

The regular monthly meeting of the Lincoln-Sagadahoc County Medical Society was held at The Ledges Inn, Wiscasset, Maine on Tuesday, May 15, 1973. There were twenty-three members and guests present.

The meeting was called to order by the President, Dr. Peter A. Evans at 8:32 p.m.; the minutes of the April meeting were read and accepted as read.

There was no old business.

Dr. Paul A. Fichtner proposed a resolution for endorsement by this county society to forbid contract between the Maine Medical Association and any branch of government to oversee quality control of medical practice. Acerbic debate followed. Two amendments were suggested, and further discussion was postponed until the end of the meeting, allowing for rewriting of the proposed resolution.

Dr. Richard C. Leck led discussion of proposed candidates for major office in M.M.A. and also of the persistent question of chiropractic.

Dr. Fichtner's proposed resolution was then reintroduced as amended, with addition of proposals to support the program of peer review in the private practice of medicine, with instructions to spread knowledge of the resolution to all members of the M.M.A. The Society voted to support the proposed resolution

and instructed that it be introduced at the Annual Meeting of the House of Delegates next month.

Dr. Evans then introduced Dr. Louis Bachrach, chairman of the program committee, who introduced Mr. Roger Fagan, audiologist at Pine Tree Society in Bath, who spoke on Clinical Audiology.

The meeting was adjourned at 10:20 p.m.

GEORGE W. BOSTWICK, M.D., *Secretary*

ANDROSCOGGIN

The meeting of the Androscoggin County Medical Association was held at Steckino's Restaurant, Saturday, May 26, 1973.

The business meeting was called to order by the President, Dr. Gilbert R. Grimes.

The secretary's report was omitted.

Dr. Kenneth Wolf was admitted to membership on recommendation of the Credentials Committee.

There being no other business, the meeting was turned over to the program chairman. The wives were guests of the physicians, and a delicious smorgasbord was enjoyed, followed by humorous entertainment presented by Mr. Murphy.

RICHARD M. SWENGEL, M.D., *Secretary*

OXFORD

The Oxford County Medical Society had its semi-annual meeting at Bethel Inn in Bethel on June 6, 1973.

The business meeting was uneventful. A film was shown on the subject of Iron Deficiency Anemia.

A dinner, with wives present, was very successful. Dr. H. Carl Amrein attended, and discussed many current issues with members.

STEPHEN B. DEWING, M.D., *Secretary*

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The Journal
of the
Maine Medical Association

Volume Sixty-four | Brunswick, Maine, September 1973 | Number 9

Some Guidelines for Surgery for Hyperparathyroidism Based on a Ten-Year Experience With Fifteen Patients at the Maine Medical Center

CLEMENT A. HIEBERT, M.D. and JEREMY R. MORTON, M.D.*

There are two unique things about surgery for hyperparathyroidism: patients are elusive and so are the glands. In the first instance, it is the patient's physician who has the opportunity, as well as, hopefully, the wit to appreciate the potential significance of such apparently unrelated symptoms as listlessness, ulcer pain, and renal colic. In the second, it is the surgeon who has the challenge of finding the Lilliputian glands, identifying the pathology, and excising the proper amount of tissue. Crucial to the success of the whole enterprise is a thoroughly reliable laboratory. This series of fifteen patients treated by the authors at the Maine Medical Center is presented and examined from the viewpoint of just what are the requirements for successful parathyroid surgery.

Presenting Complaints: According to Gordan,[1] approximately one patient in 850 unselected individuals (studied with serum calcium determinations) will have hyperparathyroidism. When only patients with suspicious symptoms are reviewed, one patient in 80 will prove to have the condition. Finally, when one examines only patients with renal calculi, one may expect to find anywhere from one in 10 to one in 30 with hyperparathyroidism.[2]

In this group of 15 patients, seven presented with renal stones, one adenoma was discovered in an otherwise asymptomatic elderly patient undergoing neck exploration for a pharyngeal diverticulum, and seven had their diagnoses, in effect, headlined on the read-out sheet of the SMA-12 Profile. In a word, with the majority of our patients, it was a matter of serendipitous stumbling. Retrospectively, however, six individuals had had complaints referable to the skeletal system, eight had mental troubles varying from unheralded crying spells to frank psychosis, two had peptic ulcers, and one pancreatitis (Table 1). Duration of symptoms was six weeks to 17 years but was less than one year in nine. Patients' ages ranged from 30 to 78. There were eight females and seven males.

TABLE 1

CLINICAL FEATURES OF HYPERPARATHYROIDISM IN 15 PATIENTS

Mental Symptoms	8
Urinary Tract Calculi	7
Bone Changes	4
Peptic Ulceration	1
Pancreatitis	1
Upper GI Complaints	2

In one of the author's Massachusetts General Hospital experience (not included in this study) a young adult female presented with a single episode of upper GI bleeding apparently from a peptic ulcer. There were no other symptoms. Initially the patient's serum calcium was normal but subsequently it proved elevated and this finding led to the discovery of a curable 1.5 centimeter parathyroid carcinoma!

Diagnostic Tests: The triad of elevated serum calcium, depressed phosphorus, and increased urinary calcium is solidly pathognomonic of the condition. By far the most important test is the

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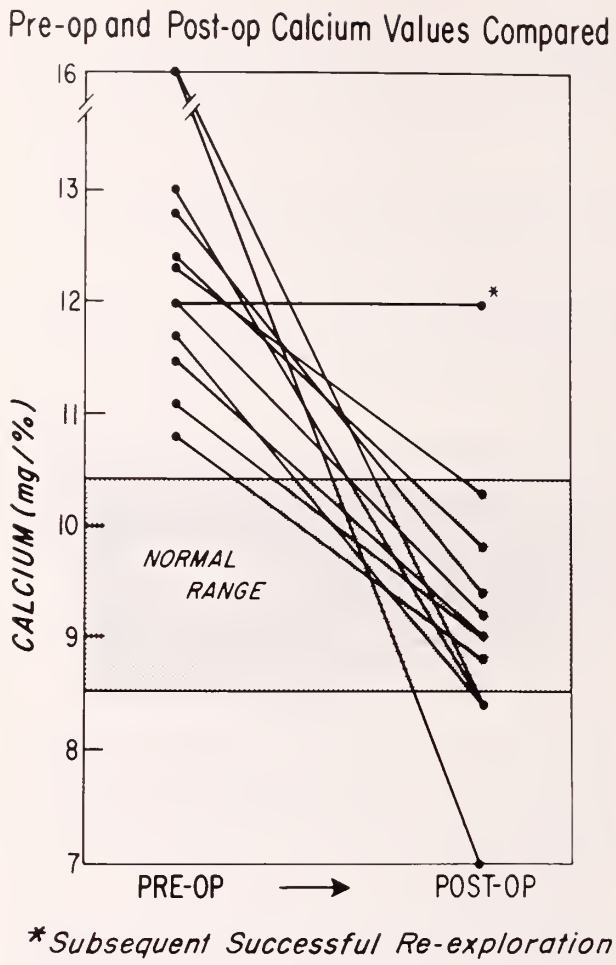

Fig. 1

serum calcium regarded by Cope as suspicious if greater than 10 mgs. % and certainly abnormal if greater than 10.5 mgs. % (assuming the serum protein is not concomitantly elevated).[2] The distribution of calcium and phosphorous values in this series is shown in Figure 1. Of four patients in whom 24-hour urine calciums were studied, two had hypercalciuria, and two did not. The latter were not operated on.

A word of caution about other causes of hypercalcemia: it is an unwelcome fact that the serum calcium may be elevated in sarcoid, multiple myeloma, prolonged skeletal immobilization, metastatic carcinoma, hypernephroma, cancer of the lung, and Vitamin D intoxication. In none of these is the serum phosphorus depressed, however, and the serum calcium generally will fall in response to a ten-day course of steroids. Renal insufficiency too may have an associated elevation of serum calcium, but here the phosphorus is normal or elevated and, in addition, there are signs of renal failure.

Other diagnostic tests include a study of bones for demineralization, particularly in the middle phalanges and the lamina dura. When all is said and done, however, it is the presence of an elevated serum calcium that makes the diagnosis and an elevated serum calcium, plus symptoms, should always lead to surgical consultation.

Surgery: The commission of the surgeon is to find the parathyroids and to excise the appropriate tissue. A theoretical ideal is to identify four glands, inasmuch as a second adenoma is always possible and also because hyperplasia, which is occasionally indistinguishable from an adenoma by frozen section, involves removing three glands and a portion of the fourth. In practice, however, when an adenoma is of sufficient size to account for the patient's elevated calcium and two other normal or hypoplastic glands have been found, statistics are on the side of the surgeon who discontinues the search after a reasonable effort to find the fourth gland has been made.

Because the parathyroids are small in comparison to other surgical quarry, because their position is so variable, and, finally, because the Creator has endowed them with protective coloration, parathyroid safaris must be planned and executed with meticulous care. This is no place for the casual operation sandwiched in the middle of a fully scheduled day. Intravenous methylene blue tagging, angiography, or parathyroid hormone assays of jugular venous blood are less important than a pair of two power optical loops, a comfortable stool, and throwing away the clock. The surgeon shoud prepare himself by reviewing the anatomy, embryology, and pathology of the glands and practice dissecting them in the postmortem room.

While it is not the purpose of this paper to detail each step of the anatomical search, several points deriving from our experience may be in order. First, the strap muscles should be divided and from hereon the dissection must be aimed toward keeping the areolar tissues unstained with blood, lest the little glands be lost in a sea of red. Judicious use of the electrocautery is helpful in eliminating blood loss through even the tiniest vessels, which should be ligated or coagulated *before* rather than after they are divided. Retraction of the thyroid is facilitated with multiple figure of 8 knotted vascular sutures passed through the gland and clasped with hemostats held by the assistant. Finally, a copy of a suitable surgical text[3] placed on an unsterile Mayo stand provides immediate reference to the areas of most profitable anatomic search.

Following the above plan, it has not been necessary to remove the thyroid gland in desperation nor have we done a single sternotomy for mediastinal exploration. Opening the sternum, in any event, in our minds is to be reserved for a later effort. Two patients indeed did have mediastinal

parathyroid adenomas: one near the thymus and the other associated with a posterior pharyngeal diverticulum. Both were retrieved through a cervical approach. Although patients are always alerted beforehand to the possibility of a second operation, this has been necessary only in one instance (see Fig. 1).

Pathology: Two patients gave a history of previous pituitary tumors. One of these had a solitary parathyroid adenoma and the other parathyroid hyperplasia. Except for one other instance of hyperplasia, the remainder had solitary adenomas. One man died of unknown causes while awaiting a decision regarding surgery and another without symptoms is being followed.

The question of whether patients with elevated serum calciums but without symptoms should have prophylactic surgery is currently being debated. Long-term studies are underway in other institutions and until they are reported operation would seem indicated only if the urinary calcium excretion is greater than 300 mgs. in 24 hours.[4] In this circumstance, concern has been expressed that the patient is passing his skeleton through his bladder and hence prophylactic surgery would seem justified.

Summary

A personal experience with 15 patients with hyperparathyroidism encountered during the last ten years at the Maine Medical Center is reviewed. More use of multiphasic screening as well as increasing awareness of the symptoms of this entity will doubtless further increase the frequency of the diagnosis. The surgeon who deals with the problem needs to prepare himself with time, meticulous technique, and, most of all, a carefully thought through operative plan.

References

1. Gordan, G. S., et al: Clinical Endocrinology of Parathyroid Hormone Excess. Recent Progr. Hormone Res. 18: 297-336, 1962.
2. Cope, O.: Hyperparathyroidism: Diagnosis and Management. A. J. Surg. 99: 394-402, 1960.
3. Cope, O.: The Parathyroids. Warren's Textbook of Surgery: W. B. Saunders Co. 581-597, 1963.
4. Cope, O.: Personal Communication.

BLUE CROSS AND OTHER INSURANCES ARE ACCEPTED

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Diagnosis and Treatment

JEREMY R. MORTON, M.D.* JAN DREWRY, M.D.** and
ROLAND G. WARE, JR., M.D.***

Recent advances in endocrinology and vascular surgery have rendered a significant number of patients with severe hypertension candidates for corrective surgery with the prospect of complete cure or marked improvement. An aggressive approach to ferret out these "surgically curable" forms of hypertension is important in order to prevent the well-known permanent sequellae of the disease.

Renovascular occlusive disease is the surgically correctable lesion most commonly encountered and will be the principal subject of this paper. The two endocrine disorders primarily characterized by hypertension, primary aldosteronism and pheochromocytoma, and parenchymal renal disease constitute the other surgically treatable causes of hypertension. Since these diseases must always be considered in evaluating a hypertensive patient, they will also be described briefly herein.

Primary Aldosteronism: In addition to hypertension, these patients frequently present with muscle weakness, polyuria, and polydipsia. Malignant hypertension is rare and eye ground changes are mild in relation to the severity of the hypertension.

Hypokalemia (unprovoked by diuretic therapy) occurs in nearly all patients with this disease and is sometimes accompanied by hypernatremia. Serum chloride is normal or increased. Normally, aldosterone secretion is stimulated by ACTH, potassium loading and hypovolemia. Hypervolemia, through suppression of the renin-angiotensin system, and potassium depletion serve as feedback inhibitors of aldosterone secretion. In primary aldosteronism, however, the hormone is autonomously secreted by an adrenal adenoma and serum aldosterone levels remain high despite hypokalemia and diminished levels of serum renin. Also serum cortisol and urinary 17-hydroxy steroids are normal.

Measurement of plasma renin activity effectively differentiates primary from secondary aldosteronism and blood pressure response to spironolactone (Aldactone®) and dexamethasone (Decadron®) aids in separating those patients with aldosterone producing adenomas from those with adrenocortical hyperplasia.[1]

Once the diagnosis is made, localization of the adenoma may be attempted by arteriography or venography or by selective renal vein determinations of aldosterone.

Following careful preoperative potassium replacement and treatment with spironolactone, resection of the adenoma can be achieved safely using either a transperitoneal or posterior approach. Since twenty-five percent of these tumors are bilateral, the abdominal approach is generally preferred.

Pheochromocytoma: Patients with pheochromocytoma may present with intermittent or sustained hypertension. Evidence of hypermetabolism such as sweating, fever, tachycardia, tremor, severe anxiety, and even paralytic ileus commonly accompanying the hypertension.

Diagnostic tests employing drugs such as histamine to stimulate release of catecholamines from the tumor or phentolamine (Regitine®) to block their pressor effects have been used. The prevalence of false positive and false negative results has limited the usefulness of these methods, however, and presently the definitive method of making the diagnosis of pheochromocytoma is by demonstrating increased secretion of catecholamines. The most reliable tests are those which measure urinary excretion of catecholamines, metanephrines, and vanillylmandelic acid (VMA). Blood catecholamine determinations may also be useful.[2]

Having made the diagnosis, control of blood pressure preoperatively with an alpha-adrenergic blocking agent such as phenoxybenzamine (Dibenzyline®) and intraoperatively with phentolamine (Regitine) is essential. Cardiac arrhythmias must be simultaneously controlled with beta-adrenergic blocking agent propranolol. Also the blood volume, which is significantly reduced by the vasoconstrictive effect of the catecholamines, should be augmented preoperatively and intraoperatively to avoid marked hypotension following removal of the tumor.

Renal Parenchymal Disease: Certain patients with hypertension due to renal parenchymal disease may be significantly benefitted by surgery. Indeed, nephrectomy may be curative in patients

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with unilateral involvement with pyelonephritis, hydronephrosis, hypoplasia, renal infarction, and certain intrarenal vascular abnormalities. With the recent advent of chronic dialysis, bilateral nephrectomy is not uncommonly performed when end stage renal failure and severe hypertension occur concomitantly. The mechanism of hypertension in these patients is again the renin-angiotensin axis and preoperative renin determinations may be of great value in predicting the response to nephrectomy.

Renal Vascular Disease: Renovascular hypertension in the broadest sense may be defined as hypertension resulting from decreased pulsatile blood flow to the kidney resulting from vascular obstruction proximal to the kidney. The obstruction may be represented by coarctation of the aorta, hypoplasia of the suprarenal aorta, or more commonly by renal artery obstruction in the form of an atherosclerotic plaque or fibromuscular hyperplasia.

The decreased arterial pulse pressure within the kidney causes release of renin by the cells of the juxtaglomerular apparatus. The renin, in turn, acts on a precursor substance from the globulin fraction of the serum to produce angiotensin I which is then converted to the potent vasopressor angiotensin II. The adrenal cortex is also stimulated by angiotensin to produce increased amounts of aldosterone resulting in sodium conservation and expansion of the blood volume.[3]

Although the rapid onset of hypertension and the presence of bruits over the flank or abdomen are suggestive of renal artery occlusive disease, these findings are uncommon and the majority of patients with renovascular hypertension cannot be separated clinically from those with essential hypertension. Therefore all young patients with significant hypertension and older patients with severe or medically resistant hypertension who are reasonable candidates for surgery should be screened for renal artery occlusive disease as well as the other previously mentioned curable forms of hypertension. Screening tests include rapid sequence intravenous pyelography, radioisotope renography, and venous renin determinations. If any of these screening tests are positive or if the hypertension is severe, renal arteriography should be performed. The most consistently satisfactory results are obtained using the retrograde Seldinger technique from the femoral artery and rapid serial filming.

Although arteriography may demonstrate an apparently significant stenosis of one or both renal arteries, the physiologic significance of the lesion must be determined before it can be incriminated as the cause of the hypertension. This may be accomplished either by measuring differentially the excretory function of each kidney, as in the Howard or Stamey Test, or by determining the concentration of renin in each renal vein by selective catheterization. The latter method is generally preferred when a dependable renin assay is available.

Having determined that one kidney, by virtue of a stenotic artery and an inordinately high renin production, is the source of the hypertension, two solutions are available: the kidney may be removed or its arterial insufficiency corrected. In cases where the involved kidney is markedly contracted or the artery irreparable, nephrectomy is the treatment of choice. In other cases, however, vascular reconstruction should be performed for two reasons: First, every effort should be made to preserve all functioning renal tissue particularly in patients with arteriosclerosis in whom progression of their disease may ultimately produce renal insufficiency. Second, if the hypertension has been present for an extended period of time, the "uninvolved" kidney may have already suffered the secondary vascular changes of hypertension, namely nephrosclerosis, with significant impairment of renal function. The kidney with the arterial stenosis will have been protected from these changes and when revascularized may be the better of the two kidneys functionally.

Reestablishing an unobstructed blood supply to the kidney may be undertaken in a variety of ways as illustrated by DeBakey's series of 627 reconstructive procedures in which 30 different techniques were employed.[4] In general, however, three basic techniques are available: endarterectomy, patch graft angioplasty, and bypass grafting. Although one of these techniques may be favored by a given surgeon for the majority of cases, the others must be held in reserve for use in specific instances, as will be illustrated in the case reports which follow.

In the majority of cases an atherosclerotic plaque is present which is discrete and involves only the first 1-2 centimeters of the renal artery. The lesion, therefore, lends itself well to endarterectomy which is often combined with patch angioplasty to further widen the diseased segment (Fig. 1).

Since exposure in this area is difficult, many surgeons prefer the use of a bypass graft from the infrarenal aorta to the midportion of the renal artery where the vessel can be exposed more easily than at its origin. This technique is also particularly applicable in arteries with a long segment of fibromuscular hyperplasia. The bypass may be constructed of autogenous saphenous vein, splenic artery, or a dacron tube graft. The use of dacron is particularly appropriate when renal artery bypass is performed in conjunction with aortic replacement surgery.

Although the renal artery lesion may be discrete, the underlying disease process is not, and these patients may exhibit a variety of other mani-

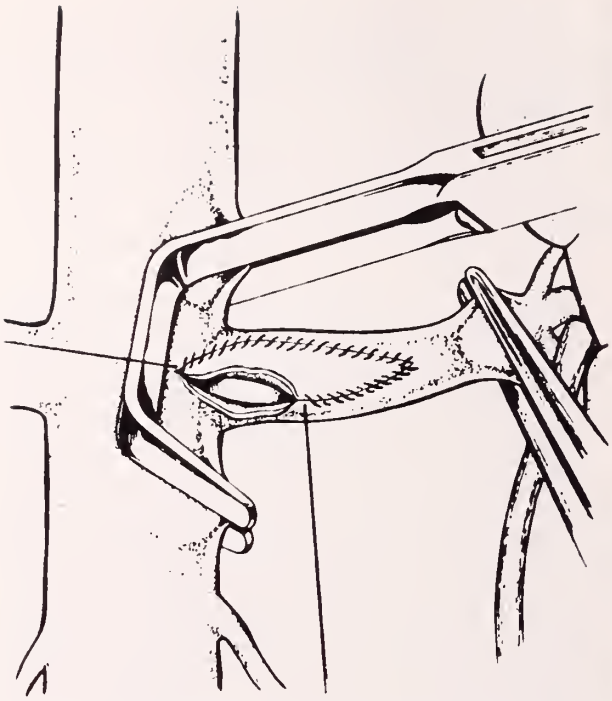

Fig. 1. Drawing illustrating technique of patch angioplasty following endarterectomy in renal artery stenosis.

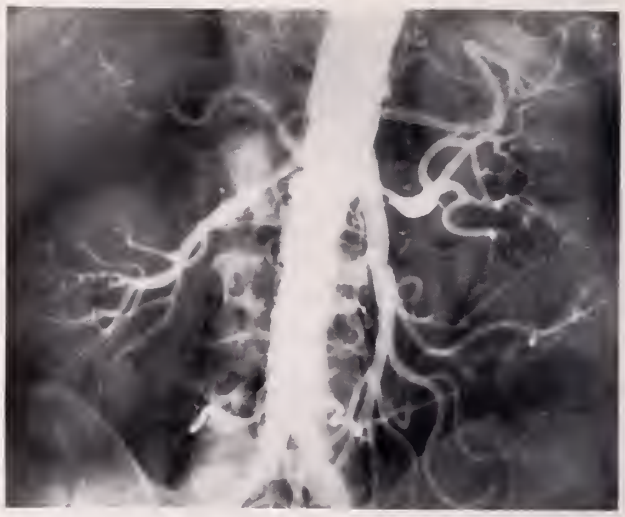

Fig. 2. Case #1 — Preoperative aortogram showing bilateral renal artery stenosis.

Fig. 3. Case #1 — Postoperative aortogram after repair of both renal arteries.

festations of arteriosclerosis which must be dealt with simultaneously. The cases which are described below illustrate several important features in the diagnosis and surgical treatment of renovascular hypertension.

Case Reports

Case #1 — C. D. (MMC #138818), a 45-year-old telephone repairman, presented with an 11-year history of progressive asymptomatic hypertension which finally became refractory to antihypertensive medications with blood pressures in the range of 210/120 mm. Hg. Physical examination was unremarkable except for multiple bruits over the abdomen and groins. All peripheral pulses were present and there were no carotid bruits. Abdominal aortogram (Fig. 2) demonstrated severe stenoses of both renal arteries and selective renal vein renin determinations revealed markedly elevated values in both renal veins. At surgery a gradient of 60 mm. Hg. was present across the stenotic renal artery lesion on the right and a gradient of 115 mm. Hg. on the left. The wall of the abdominal aorta below the level of the renal arteries was densely calcified and rigid precluding application of a vascular clamp for a bypass procedure. The aortic wall was soft in the region of the renal arteries, however, and a clamp could be applied in this area. On the left the renal artery was somewhat redundant and, in the stenotic segment, the atherosclerotic process extended through the entire thickness of the arterial wall making endarterectomy impossible. Using a partially occluding clamp on the aortic wall at the base of the left renal artery, the proximal 2 cm. of the renal artery was excised and the distal end reimplanted into the aorta at the same site. On the right, an endarterectomy was possible and was performed using the same clamping technique and a saphenous vein patch was sutured over the arteriotomy to widen the lumen further in this area. Postoperatively the patient's blood pressure fluctuated widely but remained below 180/90 without medications. Postoperative aortogram (Fig. 3) shows widely patent vessels bilaterally.

In this patient, the operative management was dictated by the pathological situation encountered and there was little room for choice.

Case #2 — J. F. (MMC #142490), an active 59-year-old businessman, presented with a one-year history of severe refractory hypertension and incapacitating headaches as well as intermittent claudication in both legs. His blood pressure remained in the range of 250/120 despite vigorous antihypertensive therapy. Physical examination was unremarkable except for slight cardiac enlargement and markedly diminished femoral pulses bilaterally. Rapid sequence intravenous pyelography revealed delayed excretion on the left. Aortogram (Fig. 4) demonstrated marked stenosis of the left renal artery, complete occlusion of the right iliac artery, and 80 percent occlusion of the left iliac artery. Selective renal vein renin determinations showed marked elevation (2270 ng. percent) on the left. At surgery, the gradient across the renal

Fig. 4. Case #2 — Preoperative aortogram showing left renal artery stenosis and aortoiliac occlusive disease.

artery lesion was 85 mm. Hg. Endarterectomy of the proximal renal artery and adjacent aorta was performed with a vein patch angioplasty as in the previous case. Following this, a bypass graft was constructed from the aorta to both external iliac arteries using a knitted dacron bifurcation graft. Postoperatively the blood pressure dropped immediately to 130/80 and remained there. The patient was totally relieved of his headaches and his claudication.

In this case, two manifestations of the atherosclerotic process required treatment simultaneously. An equally acceptable alternative method would have been to construct a dacron "side arm" graft from the aortic graft to the renal artery.

Case #3 — H. C. (MMC #137893), a 45-year-old fisherman, suffered a stroke with left-sided hemiparesis six months prior to admission. He recovered almost completely and was left with only minimal weakness in the left hand. During this episode, he was found to be severely hypertensive with a blood pressure of 240/150 and was unresponsive to medications. Examination demonstrated a mild left hemiparesis and a loud left carotid bruit with no bruit on the right. Electrocardiogram showed left ventricular hypertrophy and there was a moderate degree of albuminuria (100 mg. %). Abdominal aortogram (Fig. 5) demonstrated a long stenotic lesion of the right renal artery and an arch aortogram demonstrated complete occlusion of the right internal carotid artery and 90% stenosis of the left internal carotid artery. Biopsy of the left kidney showed moderate nephrosclerosis.

A left carotid endarterectomy was performed initially, followed in ten days by renal artery surgery. The renal artery lesion was found to have a fibrous consistency representing fibromuscular dysplasia rather than atherosclerosis and a bypass graft was performed between the aorta and the distal renal artery using a segment of autogenous saphenous vein.

Postoperatively the patient's blood pressure fluctuated between 130/80 and 170/100 mm. Hg. and he remained without symptoms or neurological change. Postoperative aortogram (Fig. 6) showed a functioning vein graft.

This patient represents two additional problems: concomitant cerebrovascular insufficiency and fibromuscular dysplasia of the renal artery. Although the patient was having

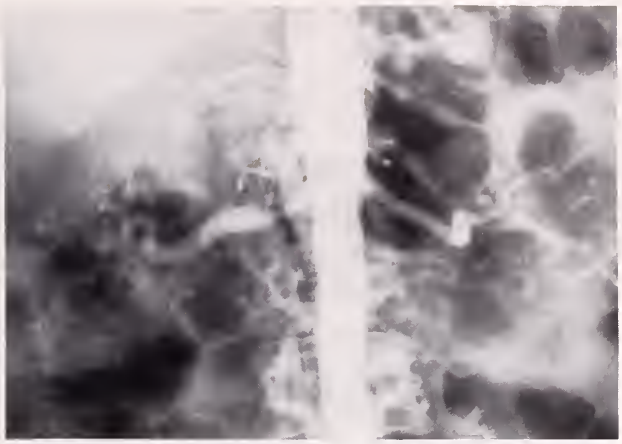

Fig. 5. Case #3 — Preoperative aortogram showing right renal artery stenosis due to fibromuscular dysplasia.

Fig. 6. Case #3 — Postoperative aortogram showing functioning aortorenal bypass graft.

no symptoms of transient cerebral ischemia, it would have been hazardous to lower the blood pressure abruptly with renal artery surgery without first insuring, insofar as possible, an adequate cerebral blood supply. Reestablishing normal cerebral circulation in the face of severe hypertension, however, is not without its own risks, as will be pointed out in the next case.

Since fibromuscular hyperplasia or dysplasia involves the entire thickness of the arterial wall, this disease is not amenable to endarterectomy and must be treated by either a long patch angioplasty or a bypass graft. In general, the bypass procedure is preferred.[5]

Case #4 — L. H. (MMC #132752), a 54-year-old housewife and factory worker, presented with a one-year history of severe hypertension and secondary congestive heart failure. She also suffered from vascular insufficiency to the lower extremities. Six years previously, she had developed an infection of the left foot which, because of associated vascular insufficiency, resulted in a below-knee amputation. Following this she did well with a prosthesis until about a year before admission when she began having intermittent claudication in the right calf and the left thigh coming on when she walked approximately one-half block.

Fig. 7. Case #4 — Preoperative aortogram showing right renal artery thrombosis, left renal artery stenosis, and aortoiliac occlusive disease.

A third problem made its appearance about three weeks prior to admission when she began having episodes of weakness in the left arm and hand occurring once or twice daily and lasting for 15 to 20 minutes.

On examination, the blood pressure was 220/140 and the pulse was 45 and regular. The heart was significantly enlarged and rales were heard in both lung bases. There were prominent bruits over both carotids. The right femoral pulse was absent, the left markedly diminished, and there were no pulses palpable below the femorals.

Chest film showed significant pulmonary vascular congestion. Intravenous pyelogram demonstrated a normal appearing kidney on the left and no excretion of contrast matrial on the right. Translumbar aortogram (Fig. 7) showed no renal artery on the right and 70 percent stenosis of the left renal artery. Also demonstrated was severe stenosis of both common and external iliac arteries. Renal vein renin assay showed marked elevation on the right (5000 mg. percent) and only slight elevation on the left. Arch aortogram demonstrated almost complete occlusion of the right internal carotid artery and 70 percent occlusion of the left internal carotid artery.

The electrocardiogram demonstrated a sinus bradycardia with left ventricular hypertrophy and strain. It was initially thought that the bradycardia was due to digitalis and this drug was discontinued. The heart rate did not change over the ensuing five days, however, and the transient cerebral ischemic attacks became more frequent. A temporary transvenous pacemaker was, therefore, inserted and a right carotid endarterectomy was performed. Despite antihypertensive medications, the patient became severely hypertensive during the first hour postoperatively and developed signs of cerebral edema with left hemiparesis. She responded dramatically to trimethaphan (Arfonad®), mannitol, and steroids with virtually complete neurological recovery in 48 hours.

During the postoperative period, the heart rate gradually increased and the pacemaker was removed. Six weeks later, abdominal exploration demonstrated an atrophic right kidney with a cord-like artery and on the left a gradient of only 20 mm. Hg. between the aorta and the left renal artery. A right nephrectomy was performed and a bypass graft constructed between the abdominal aorta and both common femoral arteries using a knitted dacron graft. The left renal artery was left alone.

Postoperatively the patient developed transient acute tubular necrosis with anuria and marked elevation of BUN and creatinine. She recovered without dialysis, however, and the blood pressure was 110/70 at the time of discharge 25 days after surgery. She has subsequently returned to work on no medications without hypertension, claudication, or congestive heart failure.

This case illustrates vividly the variety of complicating features one may encounter when treating a patient with widespread atherosclerosis. The critical importance of preoperative renin determinations is also demonstrated; in another patient with identical arteriographic findings the atrophic kidney might be producing no renin and the hypertension be due entirely to the kidney with the stenotic artery. Although the acute tubular necrosis in this case was probably aggravated by a period of unrecognized low blood volume, some degree of impaired renal function postoperatively is not uncommon in these patients and one should be prepared to provide temporary support with dialysis if the need should arise.

Comment: The serious consequences of severe diastolic hypertension and the relative ineffectiveness of medical treatment in patients whose hypertension is of renovascular origin make the accurate diagnosis of this lesion of critical importance. Since renal failure is a common cause of death in hypertensive disease, reconstructive arterial surgery should be performed in preference to nephrectomy whenever feasible. Following surgical correction of a significant renal artery stenosis, approximately 65 percent of patients are relieved completely of their hypertension and an additional 15 percent are markedly improved and become easily controllable with medications.[4,6]

The blood pressure may drop immediately postoperatively or may fall gradually over a period of weeks or months. Renal function may or may not be improved by surgery, but more importantly the steady deterioration of renal function resulting from the hypertension will be arrested.

Summary

An aggressive diagnostic approach in the seriously hypertensive patient may uncover a significant number of patients with surgically correctable disease. Although hyperaldosteronism or pheochromocytoma may be found occasionally, the majority of these patients will be found to have atherosclerotic or fibromuscular stenosis of one or both renal arteries. Surgical correction of these lesions will eliminate or significantly improve the hypertension in eighty percent of cases.

Initial work-up of hypertensive patients should include rapid sequence intravenous pyelogram, serum electrolytes, and urinary VMA excretion.

Continued on Page 214

Granulosa Cell Tumor of the Ovary in a 7-Year-Old Female With Late (12-Year) Lung Metastasis

A Case Report and Discussion

EUGENE C. MCCANN, M.D., FACOG, FACS and
JOHN ZERNER, M.D., JR. FACOG

Ovarian tumors are rare in children. One of the more unusual, granulosa cell tumor of the ovary, leads to precocious sexual development. Further, late recurrences are common with this diagnosis. Long-term follow up is mandatory with aggressive management of any reoccurrence or metastases. Because of these facts, we are reporting a case of granulosa cell tumor illustrating some of the clinical features of this particular entity.

REPORT OF A CASE

Admission #1

A 7-year-old female child was first seen at the Maine Medical Center in October of 1958 with an 18-month history of premature breast development. Otherwise, she appeared well until two days prior to admission when she was noted to have abdominal tenderness with increasing lower quadrant pain. There was no history of vaginal bleeding nor any pubic or axillary hair. Admission laboratory reports were essentially normal except for the IVP showing an ill-defined pelvic mass with mild bilateral hydronephrosis. One day following admission, increasing abdominal pain was noted; her temperature rose to 102 and she was taken to the OR for exploratory laporatomy. On exploration, approximately 2500 ccs. of straw-colored fluid was aspirated from the abdomen. A large pelvic mass measuring 17 cm. x 12 cm. x 7 cm. was found arising from the right ovary with a 1-2 mm. pinpoint opening in its anterior surface. A right salpingo-oophorectomy was performed, the entire specimen weighing 1200 grams. There was no observable abnormality of the uterus nor of the contralateral ovary. PATHOLOGIC DIAGNOSIS: GRANULOSA CELL CARCINOMA OF THE OVARY WITH CAPSULE PERFORATION (Fig. 1); NORMAL FALLOPIAN TUBE. Postoperatively, the patient did well and was discharged to be followed in the GYN Clinic.

Admission #2

Approximately one year later, September 1959, the patient now eight years of age, was admitted with vaginal bleeding and progressive breast development. Positive physical findings were: Breasts considered to be in adolescent development and tenderness to deep palpation in the right lower quadrant. Rectal examination: Cervix and uterus were felt to be somewhat enlarged; there were masses definitely felt in the right adnexal region. Laboratory studies were all within normal limits. Exploration revealed a recurrent tumor in the right adnexal region with metastatic implants on the peritoneum and on the sigmoid. Total abdominal hysterectomy and left salpingo-oophorectomy with resection of recurrent disease in the right adnexa, as well as numerous biopsies, were performed. Omental biopsies showed no evidence ot tumor nor did sigmoid node biopsy. PATHOLOGIC DIAGNOSIS: RECURRENT GRANULOSA CELL CARCINOMA. It was noted that the endometrial curettings showed proliferative stage with no adenomatous or cystic hyperplasia. Following surgery, the patient received cobalt therapy to the anterior and posterior pelvic portals including mid-abdomen which gave an estimated central pelvic and abdominal tumor dosage of 3600 R.

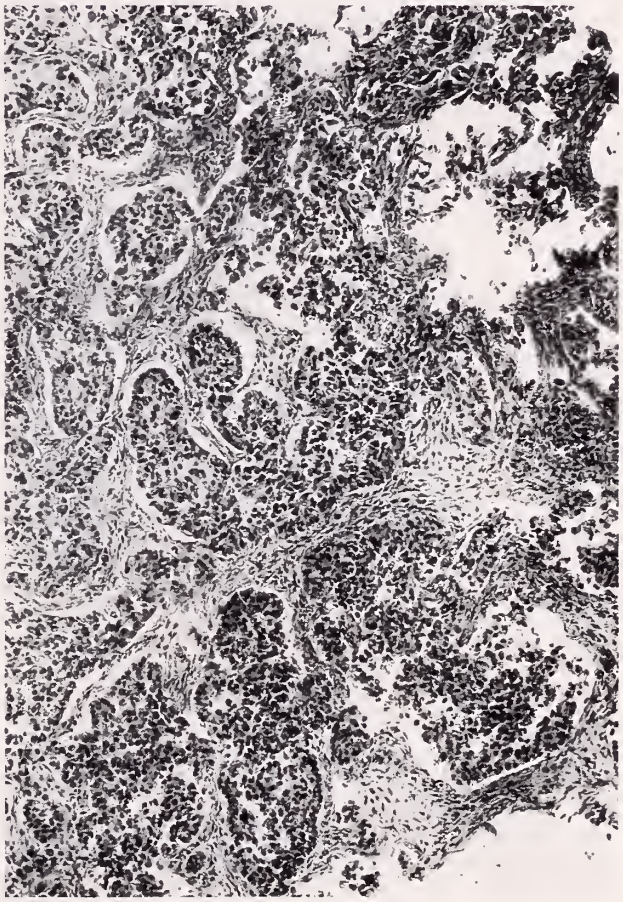

Fig. 1. Original specimen showing tumor with majority of cells being small, uniform and having rather vesicular nuclei; numerous fibrous septa around and between tumor cell groups and within some, considerable variation in size, shape and staining reaction of individual cells (100x).

Admission #3

Patient, now 17 years old, was admitted in January of 1969 with localized upper abdominal pain. Physical exam was unremarkable except for healed abdominal incisional scars and decreased breath sounds in the right chest base posteriorly. Chest x-ray showed a large round mass in the right lower chest considered to represent metastases. Laboratory studies were

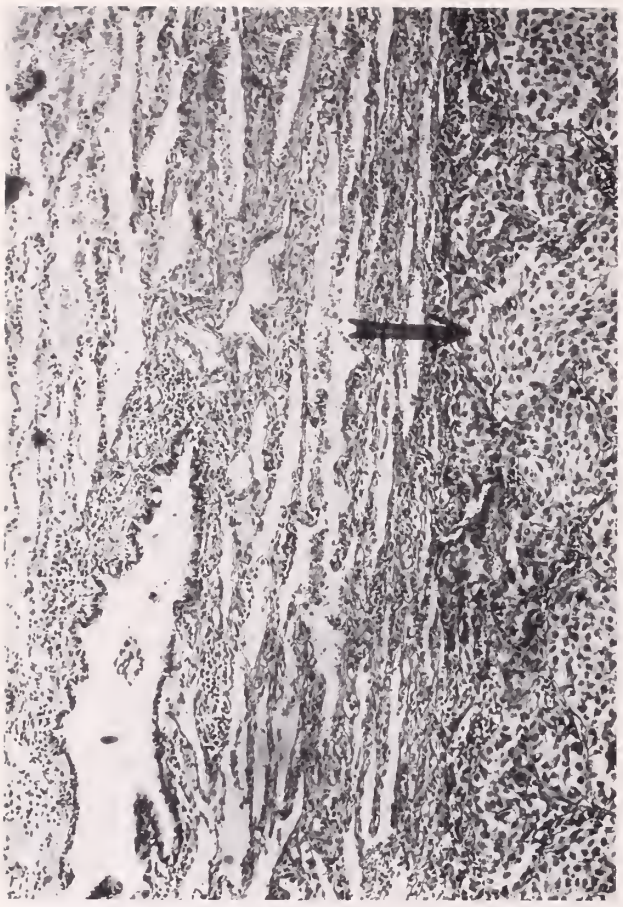

Fig. 2. Representative slide of metastatic lesion of lung 12 years after primary found; normal lung tissue on left; tumor (arrow) on right consistent with origin from granulosa cell carcinoma of ovary (100x).

essentially normal including upper GI, IVP, and barium enema. Abnormal findings were slightly elevated — alkaline phosphatase and LDH. HOSPITAL COURSE: On 2/7/69 a right lower lobectomy was performed. PATHOLOGIC DIAGNOSIS: METASTATIC CARCINOMA, CONSISTENT WITH ORIGIN OF GRANULOSA CELL TUMOR OF OVARY (Fig. 2).

From this time to the present, the patient has been essentially asymptomatic. She is receiving replacement cyclic estrogen, is married and is able to carry on sexual activities without difficulty.

The most recent examination in 1972 showed no evidence of recurrent disease. IVP, chest x-ray, and chemical profile were all within normal limits.

Discussion

Gynecological cancer in the child is extremely rare. However, ovarian tumors are the most frequent neoplasms that occur during childhood,[1] although they comprise not more than 1% of all new growths in this age group![1,13] Fortunately, the overwhelming majority of these ovarian lesions are benign, the most common types being simple follicular cysts and benign teratomas.[1,9,11] Malignancies are quite rare but, when they occur, are usually of germ-cell origins such as dysgerminomas and teratocarcinomas.[1]

Approximately 10% of childhood ovarian tumors have an endocrine function. These are potentially malignant and must be considered so until proven otherwise.[1] As mentioned by Young,[15] ovarian tumors causing sexual precocity in the young child are usually readily palpable. This is in contrast with other gonadal tumors occurring menopausally or post-menopausally which are small or barely detectable. Granulosa cell tumors, most commonly occurring after the age of 30, are very rarely found in children.[3,15,13] Scully[10] states that approximately 5% occur before puberty. However, they have been described in patients as early as 14 weeks[3] of age and furthermore, a report describes a granulosa cell tumor found in a post-mortem study of a stillborn.[8]

The tumor itself is generally unilateral and usually follows a relatively benign course.[7,15] The first manifestation of precocity secondary to excessive and premature estrogen production is usually breast development[15] followed by uterine bleeding.[3,6] Pubic and axillary hair growth are less common. The tumor itself is usually large and readily palpable abdominally.[11] The first symptom is often abdominal pain[4] frequently caused by torsion of the pedicle or rupture of the cyst itself.[9]

Treatment of the disease is based upon the initial findings at exploration. With unilateral ovarian disease limited to the capsule, conservative therapy is indicated.[1,3] A simple oophorectomy or salpingo-oophorectomy is adequate.[9] However, it is strongly urged that wedge resection of the opposite ovary be done to rule out the rare case of bilateral disease.[8] Should there be involvement of both ovaries or where tumor has penetrated the capsule, total abdominal hysterectomy and bilateral salpingo-oophorectomy is the course indicated along with radiotherapy. If there is spread beyond the pelvis, treatment is on an individual basis. Granulosa cell CA tumors are moderately radio-sensitive.[3] This tumor is noted for its recurrence both in the immediate as well as the delayed appearance. Indeed, the patient presented represents both courses. Tumor recurrence generally occurs within the next 1-4 years following initial diagnosis and treatment.

In general, a five-year survival rate in carcinoma is considered a sign of favorable prognosis. However, granulosa cell carcinomas, specifically, are noted for their late reoccurrence.[3,5,6,11] Sommers[15] has reported two incidences of recurrent ovarian granulosa cell tumors 15 and 26 years after first recognition. In a monograph by Varangot,[14*] a statement was made that ⅓ of patients without

*as quoted by Diddle, Sommers

evidence of metastases after five years could be expected to develop granulosa cell tumor metastases at a later date. This is agreed with by several others.[2,5,6,10]

Treatment of Recurrent Disease

Treatment of recurrent disease is one of surgery followed by external cobalt therapy. Should there be disease out of the pelvis, resection of the involved region is indicated if at all possible.

Summary

Thus, the patient described, presenting with reoccurrence in the pelvis, was treated with surgery plus radiotherapy. Her late metastases in the lung were treated with resection of the involved lobe.

A plea is made for the prompt evaluation of any female infant showing sudden precocious changes. Should there also be an associated pelvic mass, there is a greater than 50% chance of ovarian malignancy. These patients must be evaluated promptly and adequately. Delay in treatment is inexcusable. If the diagnosis of granulosa cell tumor is made, continued following of the patient for the remainder of her life is mandatory after initial adequate treatment. Late recurrences after 10, 15 or 20 years are common.

References

1. Acosta, A.: Gynecologic Cancer in Children. Amer. J. Obstet-Gynec. 112: 944-952, 1972.
2. Anderson, W. R.: Granulosa-Theca-Cell Tumors: Clinical and Pathologic Study. Amer. J. Obstet-Gynec. 110: 32-35, 1971.
3. Benson, R. C.: Ovarian Tumors in Childhood and Adolescence. Post Grad. Med. 50: 230-235, 1971.
4. Baker, N. J.: Malignant Tumors of the Ovary. Amer. J. Obstet-Gynec. 90: 383-387, 1964.
5. Compton, B. C.: Malignancy of Granulosa Cell Tumors. Amer. J. Obstet-Gynec. 34: 85-92, 1937.
6. Deshpande, T. V.: Granulosa Cell Tumors in 2½-Year-Old Girl with Precocious Puberty. Indian Pediat. 5: 333-337, 1968.
7. Diddle, A. W.: Granulosa and Theca-Cell Ovarian Tumors: Prognosis Cancer: 5: 215-228, 1952.
8. Norris, H. J.: Prognosis of Granulosa-Theca Tumors of the Ovary: Cancer 21: 255-263, 1968.
9. Moore, J. G.: Ovarian Tumors in Infancy, Childhood and Adolescence. Amer. J. Obstet-Gynec. 99: 913-922, 1967.
10. Morris, J. M.: Endocrine Pathology of the Ovary. C. V. Mosby Co., St. Louis, 1958.
11. Nielsen, O. V.: Ovarian Tumors in Children. Acta Obst. et Gynec. Scandinav. 47: 119-131, 1968.
12. Sommers, S. C.: Late Recurrence of Granulosa Cell Tumors. Obst. and Gynec. G: 395-398, 1955.
13. Thompson, J. P.: Ovarian and Parovarian Tumors in Infants and Children. Amer. J. Obstet-Gynec. 99: 1059-1065, 1967.
14. Varangot, J.: Les Tumeurs de la Granulosa (Folliculomes de l'Ovaire). Paris, France, Arnette, 1937.
15. Young, I. V.: Ovarian Tumors in Children and Adolescence. Post. Grad. Med. 50: 230-235, 1971.

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Indications for Coronary Arteriography

CATHEL A. MACLEOD, M.D.*

Because of the high incidence of death and disability from coronary artery disease and particularly the recent introduction of relatively low risk surgical procedures for myocardial revascularization, the role of coronary arteriography has recently assumed considerable importance. Since the procedure must be considered a major investigation, the indications for its use must involve a consideration of the relative value of the information to be obtained as compared with the possible hazards of the procedure. The primary aim must be to obtain information that will materially assist in the optimum treatment for the patient. The merits and disadvantages of the procedure need some discussion.

Coronary arteriography provides definitive information as to the presence, location and degree of severity of lesions in the visible branches of the coronary arteries. Additional information concerning the anatomical and functional state of the myocardium can be obtained when arteriography is combined with left ventriculography, either alone or with additional physiological investigations such as atrial pacing or exercise. This information may be crucial in certain patients with known or suspected coronary artery disease, but not necessarily in all.

ADVANTAGES OF CORONARY ARTERIOGRAPHY

1. The anatomical information discussed above cannot be obtained by any other method currently available.
2. The information is definitive and precise.
3. In experienced hands, the procedure is relatively safe and comparatively free from complications.

These advantages, however, presume the availability of the best cineangiographic facilities and investigators sufficiently experienced to undertake the studies with the minimum of risk and the capability of properly assessing the results. (Such facilities have recently been installed at the Maine Medical Center, and to date 88 coronary arteriograms have been performed.)

DISADVANTAGES OF CORONARY ARTERIOGRAPHY

1. RISK
   a. Mortality. The procedure, even in the best hands, does have a mortality.
   b. There is an additional morbidity from comparatively serious complications as follows:
      (I) Myocardial infarction.
      (II) Subintimal dissection of a coronary artery.
      (III) Perforation of a great vessel or cardiac chamber.
      (IV) Cerebrovascular accident from dislodgment of an atheromatous plaque or thrombus from the catheter.
      (V) Ventricular fibrillation.
   c. Less serious complications relating to the site of the incision in the case of the brachial arteriotomy technique or the arterial puncture site in the case of the percutaneous technique.
      (I) Thrombosis of either radial or femoral artery.
      (II) Infection.
      (III) Hemorrhage or hematoma.
2. EXPENSE. Besides the use of the usual hospital facilities, the procedure involves very sophisticated, expensive equipment and highly skilled personnel.

Risks

1. Mortality. Considerable variation exists in reported series. Cleveland Clinic (20,000 cases) reports less than one per thousand deaths.[1] Other series, however, show mortalities up to 2%. That seems unacceptably high.[2] There is clearly some relation to two aspects, the experience of the operator and the technique employed. Most of the higher mortality reports have related to the percutaneous femoral technique, although in Judkins' hands the reported incidence is low, 2 deaths in 4,500 cases.[3] In a recent report of a small series where both techniques were used, mortality from the percutaneous technique, 6 in 250 cases, compared with Sones technique, 0 in 111 cases.[4] (The author's experience is 2 deaths in over 1,500 cases using Sones technique, 0.6%.)
2. Myocardial infarction — variously reported as between 0.3 — 0.9% in larger series. (Author's experience 0.6%.)
3. Dissection of a coronary artery has been reported particularly with the percutaneous technique, although recent modifications of the catheter may have reduced this incidence. (Author — no cases.)
4. Embolic and thrombotic complications. The incidence of embolic complications is undoubtedly higher with the percutaneous technique and variously related to thrombogenic properties of the catheters together with the use of multiple catheters and guide wires. Thrombosis of the brachial artery

Continued on Page 205

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(Sones technique) has similarly varied from 0.5 — 8.5%, but is rarely accompanied by permanently disabling symptoms. The percutaneous femoral technique is associated with a similar incidence with potentially graver consequences. In many instances, thrombectomy and reconstructive operations can mitigate the effects.

It must be pointed out that although the reported incidence of complications is much higher with the percutaneous technique, a most important factor may be that the technique is easy to use and is often, therefore, employed by less experienced investigators. The author personally favors the brachial arteriotomy technique except in rare instances.

General Indications for Coronary Arteriography

1. Diagnosis. Under certain circumstances, when the presence of coronary artery disease is truly in doubt, AND A DEFINITIVE DIAGNOSIS MAY MATERIALLY ALTER THE COURSE OF TREATMENT.

A. Unexplained left heart failure. Coronary arteriography may differentiate between a surgically approachable ventricular aneurysm and cardiomyopathy.

B. Valvular disease with symptoms or signs at variance with the clinical estimate of severity. A study combined with routine catheterization may materially influence the choice of treatment.

C. Suspected coronary artery disease. In some instances, the diagnosis is in doubt and may be resolved only by arteriography. It must be emphasized, however, that clinical curiosity is not an adequate indication. In many such patients, no surgical intervention would be contemplated and treatment will be uninfluenced by a definite diagnosis, e.g., a single episode of pain at rest without ECG or enzyme changes may lead to hospital admission for suspected coronary artery disease. The initial management of the patient will be directed to the reduction of risk factors whether coronary disease exists or not. Subsequent management will depend upon subsequent symptoms. On the other hand, certain patients with atypical chest pain presenting diagnostic difficulties may present repeatedly with symptoms prompting admission to a coronary care unit. In some instances, multi-level exercise testing may resolve the problem, but in others, coronary arteriography may be necessary, either for the patient's reassurance or to enable the physician to manage the case with more certainty.

2. Research. Much recent knowledge of coronary artery disease is derived from coronary arteriography. Much more yet remains to be established, particularly in relation to the prognostic implications of various categories of coronary and ventricular lesions, and evaluation of therapeutic interventions can only be derived from such studies. This should not be interpreted to mean that casual investigation of patients should be undertaken under this guise. Legitimate cause exists only where a well defined protocol exists in an institution properly equipped to complete a study that can be realistically expected to provide a conclusion of value.

3. Selection of Treatment. The majority of coronary arteriograms will be performed in patients with clearly established coronary artery disease, particularly where surgical therapy is contemplated. Recent reports have established that coronary artery by-pass graft surgery can be carried out with sufficiently low mortality to justify its use in the treatment of certain categories of patients and, indeed, to suggest that it may be the treatment of choice in certain instances.[17-21] Similarly, ventricular aneurysm resection has been shown to be feasible with an acceptable mortality as the treatment of choice for certain categories of patients with this complication. Not all patients with coronary artery disease, however, are suitable candidates for surgery, and in many instances this determination can and should be made without coronary arteriography. In others, the decision as to suitability for surgery can only be made after appropriate anatomical and physiological studies have been performed. Certainly, all patients selected for coronary artery or ventricular resection surgery should be studied before such procedures are undertaken.

Selection of surgery as a therapeutic possibility, therefore, demands some appreciation of the anticipated and observed results of such intervention as compared with the results of more traditional methods of management. This involves some appreciation of the natural history of coronary artery disease and the reported results of surgical interventions.

Aims for Surgery

1. Improvement in life expectancy.
2. Reduction in potentially life threatening complications of coronary artery disease.
   - A. Myocardial infarction
   - B. Heart Failure
   - C. Arrhythmias
3. Relief of symptoms.

Natural History of Coronary Artery Disease

Numerous studies have indicated variable survival figures for patient suffering from coronary artery disease. Most studies have indicated that the life expectancy is similar whether the initial event is angina pectoris or myocardial infarction.

These studies indicate that while coronary disease diminishes life expectancy, appreciable proportions of afflicted patients survive for 5 years and 10 years from the onset of their symptoms. The two most recent studies listed above suggest a cur-

TABLE 1

SURVIVAL IN CORONARY ARTERY DISEASE (MALES)

Author	No. of Cases	Presenting Symptom	% Surviving 5 Yrs.	% Surviving 10 Yrs.	Annual Mortality Rate	% New MI 1 Yr.
Block (1952)[5]	6882	ANGINA	58	37	6	
Richards (1956)[6]	456	ANGINA	64	41	6	
Frank (1968)[7]	275	ANGINA	—	—	4 (2.5 yrs.)	4
Kannel (1972)[8]	119	ANGINA	85	58	4	
Beard (1960)[9]	427	MI	69		6 (5 yrs.)	
Pell (1964)[10]	932	MI	74		5 (5 yrs.	
Richards (1956)	162	MI	50	31	7	
Shapiro (1968)[11]	564	MI	84	(4.8 yrs.)	<4	6

(MI — Myocardial Infarction)

rent mortality rate of approximately 4% per year. This is somewhat lower than in earlier reported series, but still represents four times the risk of death of unafflicted individuals in the same studies. Similarly, the HIP study[7] shows a 5% per year incidence of new myocardial infarction in patients without previous infarction and 6% per year in patients with a previous infarction.

These studies represent total afflicted populations and considerably less information is available concerning groups particularly at risk. Such special groups might be particular candidates for a new therapeutic approach.

1. Coronary insufficiency without myocardial infarction (accelerated coronary syndrome). Despite the prevalence of this syndrome, comparatively little accurate information is available. Two recent studies,[12,13] however, suggest that while the initial in-hospital mortality is small, the risk of dying in the 18 months following the onset of the syndrome is markedly increased.

TABLE 2

PROGNOSIS OF ACUTE CORONARY INSUFFICIENCY

Author	No. of Cases	Hospital Deaths	Hospital MI	1 Yr. Survival	Died 18 Months
DeSanctis MGH[12]	100	1	6%	85%	26%
Harrison Stanford[13]	170	1	4%	90%	15%

2. The HIP study[7] also confirms previous reports[5,6] that the mortality is unfavorably influenced by hypertension, presence of an abnormal ECG, and smoking and, in particular, identifies a group where the former two abnormalities are present and the mortality rate of 11.5% per year is 10 times that of those without those abnormalities, and forms an especially high risk group with a predicted mortality resembling that of coronary insufficiency.

3. The prognostic implications of lesions demonstrated at coronary arteriography is poorly documented, but a recent study by Webster, et al[14] indicates sharply increased mortality rates for patients with three major vessels affected (10% per year) leaving only 30% survivors after 7 years. Two vessel disease (6.8% per year) shows a higher incidence than average, and with a single vessel affected, the mortality for anterior descending lesions of 4% per year is significantly greater than that for isolated lesions of the right coronary artery or circumflex branch, which is 1.8% per year and differs little from that of the general population. Similarly, Ross [15] reported no cardiac deaths in 32 patients with single vessel disease followed for an average of 4.5 years.

4. Congestive Heart Failure. The well recognized disadvantage of this complication is confirmed by many studies. The HIP study indicates a mortality rate of over 7% per year and 3 times that of men without failure. Incidence of myocardial infarction of 13% in patients with angina is similarly 3 times that of those without failure.

Two particular groups with this syndrome merit special mention.

A. Ventricular aneurysm. Although mortality figures for all cases with this complication are difficult to determine, life expectancy for clinically recognized aneurysms rarely exceeds 5 years, with the majority dying within 3 years. The true incidence of aneurysm demonstrated angiographically, however, is considerably greater than the incidence recognized clinically. (Personal observation, 25% of all studied cases. Autopsy series 20%, Schlichter and Hallestein[16]). It is apparent that many such aneurysms are tolerated with symptoms of only moderate exertional limitation of considerable periods of time. With the onset of complications, however, life expectancy is materially shortened. Congestive heart failure becomes intractable to medical therapy; systemic embolism and ventricular arrhythmias also threaten sudden death.

TABLE 3

MORTALITY OF CORONARY BY-PASS GRAFT SURGERY

Author	No. of Cases	Early Mortality	Late Mortality (2 yrs.)
Favaloro[17]	1600	4.5	
Cooley[18]	1105	6.6	
Morris[19]	480	6.2	
Johnson[20]	310	14.0	6.0
Ankeney[21]	197	4.5	3.5

B. Papillary muscle dysfunction leading to heart failure is a significant cause of disability and early death.

Results of Surgery

I. Coronary By-Pass Graft Surgery

1. Survival. These figures from major institutions suggest that while these figures are comparatively low for major cardiac operations, in effect, an operation carries with it the same risk of dying as does one year without operation and that at least up to 2 or 3 years, little improvement in life expectancy ALONE accrues from operation. It must be pointed out, however, that these figures include the groups' earlier experience and all report improved mortality rates in the later cases. Favaloro, et al recently reported mortality rates less than 1% for single or double graft operations. Similarly, two other reports on favorable groups Ankeney (Cleveland) 2.2% mortality for one or two grafts in 184 patients with a two-year total survival of 94%. Johnson (Milwaukee) for another favorable group reports a similar 94% two-year survival.

UNFAVORABLE GROUPS. a) Patients in heart failure clearly carry such a high mortality (Spencer[22]) as to suggest that such patients are unsuitable for by-pass graft surgery. b) Similarly, marked impairment of ventricular function without failure has such an adverse mortality as to suggest the same conclusion (Johnson 30%). c) The presence of distal obstructive lesions beyond the proposed site of graft insertion is associated with such a high graft failure rate as to contraindicate the procedure.

2. Myocardial Infarction. Several series carefully studied from reputable institutions suggest myocardial infarction in relation to surgery is by no means unusual.

Table 4

Coronary Surgery — Incidence of Infarction

Author	*N. of Cases*	*% MI*
Kansal[23]	151	9.9
Friedburg[24]	193	7.8
Brewer[25]	252	20.2
Morris[19]	480	8.2

In the above series, the patency of the grafts appears closely related to these complications. Most of the above studies report on restudy up to six months postoperatively an incidence of graft occlusion varying between 10 and 15%. While the long-term patency of such grafts is not yet known, current indications do suggest that between 6 months and 3 years the further attrition of grafts is comparatively uncommon and, indeed, rare except in those grafts that have been demonstrated to be unsatisfactory when studied in the early postoperative phase.

3. Relief of Symptoms. Virtually all large series have shown, however, a significantly more encouraging picture in respect of relief of angina. Most have shown complete relief of clinical angina in 50-70% of patients with substantial improvement to be expected in up to 80-90%. These findings are so universal as to encourage belief in their reliability. Objective measurements of improvement in myocardial ischemia have been limited but a significant number of small series have demonstrated objective improvement by multilevel stress testing, lactate extraction and other methods.

On the other hand, where the symptoms have indicated "power failure," while sporadic improvements have been reported, the general results have been poor with little or no improvement and even deterioration following apparently anatomically successful surgery.

II. Surgery for Coronary Insufficiency

Currently reported series for survival and symptomatic improvement following surgery in this group are few. Larger series, however, suggest that the immediate mortality is not significantly greater than that of patients with chronic angina (Johnson, 80 cases — 2 deaths, Effler 65 cases — 5 deaths).[26,27] It has been suggested that the presence of the syndrome is sufficient indication in order to prevent myocardial infarction. However, both medically treated series reported above suggest that, in fact, the incidence of immediate myocardial infarction in this group is small (6%). Immediate intervention in the entire group, therefore, would appear to be unjustified. It is possible, however, that where symptoms persist for 72 hours following admission and intensive therapy in a coronary care unit, that in view of these results, intervention might be justified in this group.

III. Surgery for Acute Myocardial Infarction

This has consisted of by-pass graft surgery performed with the aim of limiting the extent of necrosis by improving blood supply to the ischemic zone, sometimes combined with infarct resection. Mortality rates of 50% for surgery attempted in the first week following infarction would appear to outweigh any proposed benefit.[18]

IV. Surgery for Early Complications of Myocardial Infarction

Sporadic successful reports of surgery for acute repair of ventricular septal defect or papillary muscle rupture complicating myocardial infarction have been reported. The general experience to date has been disappointing. It is conceivable that the addition of intra-aortic balloon assisted circulation might improve the prospect in those that now fail to survive. Patients surviving the acute episode

have been successfully repaired in small series and this operation would appear justifiable since few survive one year untreated.

V. Surgery for Later Complications of Myocardial Infarction

1. Ventricular aneurysm. If the residual myocardium shows evidence of good function and relatively intact blood supply, mortality for aneurysm resection is approximately 10%.[28] This risk would appear acceptable if the aneurysm is complicated by the presence of heart failure resistant to medical therapy, systemic embolism or recurrent ventricular arrhythmias. In the presence of angina, resection combined with by-pass grafting also appears an acceptable procedure, but in the absence of these complications, resection is of questionable value.

2. Papillary muscle dysfunction with chronic mitral insufficiency sufficient to produce heart failure may similarly benefit from mitral valve repair combined with by-pass grafting, provided remaining myocardial function is adequate. If severe generalized myocardial dysfunction exists, however, the mortality from that complication would logically appear even greater if valve surgery were added to by-pass grafting.

Conclusions Regarding Coronary By-Pass Graft Surgery

1. Relief of angina can be anticipated in 70-95% of surviving patients.
2. Improvement in symptoms from myocardial impairment is minimal in most instances.
3. Evidence for improved life expectancy in the short term has not been demonstrated and in the long term is currently not available.
4. Evidence for reduction of the incidence of myocardial infarction is also lacking.
5. The incidence of myocardial infarction immediately following surgery is actually higher than the anticipated 1-year incidence in patients without previous infarction and approximately the same as anticipated in those with a previous infarction.
6. Operative mortality for patients with favorable lesions and good left ventricular function may be as low as 1-2% and is, therefore, an acceptable procedure if patients suffer from significant angina.
7. Operative mortality is prohibitive in patients with severe left ventricular dysfunction or heart failure.
8. Operative mortality for acute coronary insufficiency appears acceptable in patients having recovered from acute coronary insufficiency.
9. Operative mortality is acceptable for patients with continuing coronary insufficiency 72 hours after institution of intensive care therapy.
10. Operative mortality is acceptable for patients with life-threatening complications in the presence of a ventricular aneurysm.

Discussion

Patients with angina, therefore, appear candidates for surgery. It is as yet not demonstrated that mortality differs in patients with mild or severe angina. On the other hand, in the absence of any demonstration of improvement in life expectancy, operation for this cause must be regarded as primarily symptomatic treatment aiming to improve the quality of survival. Consequently, if this symptom can be readily controlled by other symptomatic treatment at the present time, it would not appear that operation was warranted. Clearly, the degree of angina indicating need for surgery will vary with the individual needs of the patient and also with the judgement of the physician. The final arbiter in many such cases must be the cardiologist undertaking coronary arteriography, who may be in a position to weigh the severity of demonstrated lesions and the quality of myocardial function against the degree of disability. It must be recognized, however, that in general, symptoms represent a late stage in coronary artery disease and that any symptomatic patient is liable to show at least one, and more probably, two or three severely obstructing lesions.

It is also recognized, however, that angina is a very subjective symptom and some assistance in the final decision can often be achieved with other objective demonstrations of myocardial ischemia such as stress testing.

In a few patients without current angina pectoris, repetitive incidents of myocardial necrosis may separate them from the more usual patient whose initial infarct may be followed by a long period of freedom from symptoms. This group might merit further investigation by stress testing and coronary arteriography, even in the absence of current symptoms.

Another group who might be considered to be at special or unusual risk might be those patients under the age of 40 who have already suffered a myocardial infarction and in whom coronary arteriography may reveal lesions particularly suitable to surgical correction with much lower than average risks of mortality or subsequent graft failure.

Patients with myocardial infarction currently asymptomatic. It is a well established surgical maxim that it is difficult to improve upon the asymptomatic patient. In the absence of demonstrated benefits of intervention, graft surgery would not appear indicated in this group. Two groups at special risks have been proposed as possible candidates for surgery even without symptoms. The first such category are those cases with obstructive lesions affecting the main trunk of LCA. It is clear that this group does, indeed, have the higher risk of sudden death. However, operative reports have also suggested a significantly increased mortality in

this group which represents only 5% of studied cases and are rarely, if ever, asymptomatic. Similarly, although the majority of cases with myocardial infarction have three vessel disease, despite the small series mentioned above, insufficient documentation exists as to the relative life expectancy of this group without symptoms as compared with the general cohort of patients with coronary heart disease.

Secondary to this view, the timing of study in relation to a recent myocardial infarction is very relevant. In a patient with previous angina following a recent infarction the subsequent symptomatic state may be significantly altered. Angina may be more severe, less severe, or even disappear. Therefore, it is my view that coronary arteriography should not immediately follow a recent infarction. There is good evidence to suggest that ordinarily there is just slightly higher risk of study in the first month following infarction. In addition, however, in order to assess the suitability of the patient for surgical therapy, the current status of symptoms must be assessed and this can only be done after the patient has returned to his normal activity. Consequently, in general, coronary arteriography should be delayed for a period of 2 or 3 months following a myocardial infarction unless it has previously become evident that disabling symptoms still exist. Similarly, in a patient whose first symptom is a myocardial infarction the same period should be permitted to elapse in order to evaluate the patient's symptomatic state.

In general, coronary arteriography for patients being considered for surgery should be carried out in close proximity to the proposed procedure. If a decision to postpone the surgery until a later date is made, the investigation should similarly be postponed. Failure to do so may result in the necessity for a second study. If an interval of any length elapses, either the state of the arteries or of the myocardium may have changed and have rendered invalid the previous assessments of the type and extent of surgery to be performed. Similarly, the judgement as to when operation should be attempted is primarily on the basis of the above mentioned criteria and such decision should be made before investigation and not subsequently.

Relevance to Current Information

It is recognized that the above discussion represents a personal view which is somewhat conservative in its general outlook. Nevertheless, the author feels that it is probably representative of the view held by the majority of clinical cardiologists including the majority of those undertaking coronary arteriography. It is recognized that it is markedly at variance with the views proposed by some prominent workers in this field, particularly those who favor coronary arteriography and by-pass graft surgery in all patients with demonstrable coronary obstructive lesions. The author believes that that view is not currently supported by available evidence. Should that evidence become available, namely, that studies demonstrate that either the incidence of myocardial infarction is reduced or that life expectancy is significantly increased in patients treated with by-pass graft surgery irrespective of symptoms, the author would most happily change his current views. Certainly, that would represent a great advance in the benefits we could currently make available to the large number of patients who now face serious restrictions of life expectancy and disability.

Until that time, however, it is my belief that the following questions should be considered before undertaking coronary arteriography.

1. Can the information appropriate to the patient's situation be obtained by coronary arteriography?
2. Is coronary arteriography the appropriate investigation or can the information be obtained by less extensive or less hazardous methods?
3. Will the information be used? Will the patient's treatment be changed in some way as a result of the findings or will the treatment be uninfluenced by the findings?
4. Is this the appropriate time to seek the information?

Specific Indications for Coronary Arteriography

1. Patients with angina significantly interfering with normal activity.
2. Patients with accelerated angina not earlier than one month following the onset of the syndrome.
3. Patients with accelerated angina persisting more than 72 hours following admission.
4. Patients with repetitive infarction without interval angina but with objective evidence of continual myocardial ischemia on exertion.
5. Young persons below 40 with any evidence of myocardial ischemia.
6. Patients with frequent atypical chest pain whose management constitutes a problem.
7. Patients with unexplained left ventricular failure.
8. Patients with valvular disease with disproportionate cardiac disability.
9. Patients with suspected ventricular aneurysm, symptoms of intractable heart failure, systemic emboli or recurrent arrhythmias.
10. Patients with suspected coronary disease in certain specific occupations where absolute diagnosis is a private or public necessity (e.g., airline pilots, Presidential candidates).
11. Postoperative evaluation of surgically treated patients.

Relative Contraindications to Coronary Arteriography

1. Currently asymptomatic patients with remote myocardial infarction (at least over 45 years of age).
2. Patients with angina easily controlled by nitrites or with infrequent symptoms.
3. Patients with suspected ventricular aneurysm in the absence of angina, heart failure, emboli or arrhythmias.
4. Cardiogenic shock.
5. Digitalis toxicity or electrolyte imbalance.
6. Patients in heart failure not optimally controlled.
7. Less than 4 weeks following documented myocardial infarction.

Summary

This paper briefly reviews the natural history of coronary artery disease with and without surgical interventions.

The relative risks of the procedure are briefly reviewed and the current indications for consideration of surgery for coronary heart disease and for coronary arteriography are outlined.

References

1. Sheldon, W. C.: "Cine Coronary Arteriography," Surg. Clin. N. A. 50: 1015, 1971.
2. Selzer, A., Anderson, W. L., March H. W.: "Indications for Coronary Arteriography," Calif. Med. 115: 1, 1971.
3. Judkins, M. P.: "Percutaneous Transfemoral Coronary Arteriography," Radiol. Clin. N. A. 6: 467, 1968.
4. Petch, M. C., Sutton, R., Jefferson, J. E.: "Safety of Coronary Arteriography," Brit. H. J. 35: 377, 1973.
5. Block, W. J., Crumpacher, E. L., Dry, T. L., Gage, R. P.: "Prognosis of Angina Pectoris," J.A.M.A. 150: 259, 1952.
6. Richards, D. W., Bland, E. F., White, P. D.: "Twenty-five Year Follow-up of 456 Cases With Angina Pectoris," J. Chron. Dis. 4: 423, 1956.
7. Weinblatte, Frank C. S., Shapiro, S., Sager, R. V.: "Prognostic Factors in Angina Pectoris," J. Chron. Dis. 21: 231, 1968.
8. Kannel, W. B., Feinleib, M.: "Natural History of Angina Pectoris," A. J. Cardiol. 29: 154, 1972.
9. Beard, O. W., Hipp, H. R., Robbins, M., Taylor, J. S., Ebert, R. V., Beran, L. E.: "Initial Myocardial Infarction Among 503 Veterans," A. J. Med. 28: 871, 1960.
10. Pell, S., D'Alonzo, C. A.: "Mortality and Five Year Survival of Employed Men," N. E. J. M. 270: 915, 1964.
11. Shapiro, S., Weinblatt, E., Frank, C. W., Sager, R. V.: "Prognosis of Men After First Myocardial Infarction," A. J. Pub. Health 58: 1329, 1968.
12. Krauss, K. R., Hulter, A. M., De Sanctis, R. W.: "Acute Coronary Insufficiency," Circ. 45 (Suppl. I) I-66, 1972.
13. Lopes, M. G., Spivack, A. P., Harrison, D. C., Schroeder, J. S.: "Prognosis of Non-infarction Coronary Care Unit Patients," A. J. Cardiol. 31, 144, 1973.
14. Moberg, C. H., Webster, J. S., Sones, F. M.: "Natural History of Severe Proximal Coronary Disease," A. J. Cardiol. 29: 282, 1972.
15. Freisinger, G. C., Page, E. E., Ross, R. S.: "Prognostic Significance of Coronary Arteriography," Trans. Assoc. Amer. Phys. 83: 78, 1970.
16. Schlichter, J., Hellerstein, H. K., Katz, L. N.: "Aneurysm of the Heart," Medicine 33: 43, 1954.
17. Favaloro, R. G.: "Direct and Indirect Coronary Surgery," Circ. 46: 1197, 1972.
18. Dawson, J. T., Hall, R. J., Hallman, G. L., Cooley, D. A.: "Mortality of Coronary Artery By-pass After Previous Myocardial Infarction," A. J. Cardiol. 31: 128, 1973.
19. Morris, G. C., et al: "Follow Up Results of Distal Coronary Artery By-pass for Ischemic Heart Disease," A. J. Cardiol. 29:180, 1972.
20. Manley, J. C., Johnson, W. D.: "Effects of Surgery on Angina and Myocardial Function," Circ. 46: 1208, 1972.
21. Ankeney, J.: Personal Communications, University Hospitals, Cleveland.
22. Spencer, F. C., Green, G. E., Tice, D. A., et al: "Coronary Artery By-pass Grafts for Congestive Heart Failure," J. Thor. Cardiovasc. Surg. 62: 529, 1971.
23. Kansal, S., Reitman, D., Sheffield, L. T.: "Acute Myocardial Injury Following Aortocoronary By-pass Surgery," A. J. Cardiol. 31: 140, 1973.
24. Friedberg, H. D., Zeff, H. J., Silberman, R. E., Tector, A. J.: "Myocardial Infarction Following Coronary Surgery," A. J. Cardiol. 31: 132, 1973.
25. Brewer, D. L., Bilbro, R. H., Bartel, A. G.: "Myocardial Infarction As A Complication of Coronary By-pass Surgery," Circ. 47: 58, 1973.
26. Flemma, R. J., Johnson, W. D., Tector, A.: "Surgical Treatment of Pre-infarction Angina," Arch. Int. Med. 129: 828, 1972.
27. Cheanvechai, C., Effler, D. B., et al: "Emergency Myocardial Revascularization," A. J. Cardiol. 31: 125, 1973.
28. Favaloro, R. G., Effler, D. B., Grover, L. K., et al: "Ventricular Aneurysm," Ann. Thor. Surg., 6: 227, 1968.

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Congenital Diaphragmatic Hernia

MARTIN J. BELL, M.D.*

In the light of recent advances in pediatric surgical technique and in the management of neonates with severe surgical problems, it is warranted to review a recent experience with infants having congenital diaphragmatic hernia.

This defect is reported to occur in from 1 in 7000 to 0.8 in 10,000 live births.[1,2,3] In Maine, diaphragmatic hernia ranks high as a lethal congenital anomaly. A preliminary review of neonatal mortality in the State of Maine during the five-year period from 1967 through 1971 uncovered twenty infants whose death was due to this defect. Expressed otherwise, 8.8% of all babies dying in the first month of life because of a structural anomaly died because of congenital diaphragmatic hernia. Only cardiac defects, abnormalities of the gastrointestinal tract, and anencephaly were more frequent causes of death in this group.

Between December 1968, and February 1973, nine infants with congenital diaphragmatic hernia have been treated at the Maine Medical Center. They form the basis of this report.

PATIENT DATA

Birth Weight: The range was from 2523 grams to 3799 grams, with a mean birth weight of 3289 grams.

Sex: There were 6 male and 3 female infants.

Onset of Symptoms: Eight of the patients developed symptoms within the first 24 hours of life. One child became symptomatic in the ninth month of life.

Presenting Symptoms: The predominant symptom was respiratory distress, present in varying severity in eight of the nine patients. One patient, who was admitted at age nine months, presented with abdominal pain and vomiting.

Physical Findings: With the exception of the one patient who presented with symptoms referable to the intestinal tract, the remaining patients all revealed physical findings referable to the respiratory system.

Dyspnea	8	Bowel sounds in chest	4
Dextrocardia	7	Thoracic asymmetry	3
Cyanosis	6	Scaphoid abdomen	3
Decreased ipsilateral breath sounds	5	Apnea	1
		Bradycardia	1

Radiographic Findings: Plane films were diagnostic in all eight newborn infants, demonstrating gas-filled viscera within the chest (Fig. 1). In only one baby was an upper gastrointestinal series done, and this was confirmatory. In the nine-month-old infant with gastrointestinal symptoms, barium enema and upper gastrointestinal series were done, showing malrotation of the intestine. Here too, the chest film showed evidence of gas-filled viscera in the chest.

Associated Abnormalities: Malrotation of the midgut was present in six patients. In the remaining three, no mention was made of the presence or absence of this associated abnormality.

The following unrelated abnormalities were present in one instance each:

Communicating hydrocephalus
Patent ductus arteriosus
Polydactyly
Sacral skin tag
Accessory spleen

Operative Technique: Eight patients underwent operation.

Incision — abdominal	7	
left paramedian	4	
left subcostal	2	
midline	1	
thoracic	1	
Interrupted nonabsorbable suture repair of defect		8
Chest tube placed		5
Gastrostomy placed		2
Ventral hernia created		1

Operative Findings: All hernias were present on the left side (Fig. 2).

Left posterolateral defect (Bochdalek)	8
Sac present	1
Eventration	1

Ipsilateral pulmonary hypoplasia was noted in six patients at operation, and in one patient, pulmonary hypoplasia was noted at autopsy (Fig. 3). In two patients, no mention was made of the appearance of the lung.

Results: One infant was in severe respiratory distress shortly after birth, and died within three hours, before operation could be undertaken. Eight patients underwent operation, with repair of the abnormal diaphragm. Of these, one patient, who did well initially, developed a left (ipsilateral) tension pneumothorax on the day after operation. Despite placement of a chest tube for decompression, severe respiratory distress persisted, with hypoxia and respiratory and metabolic acidosis, to which the patient succumbed. The mortality for the entire group was 22%, and for the newborns, 25%.

At the age of seven weeks, one patient had a transient episode of partial small bowel obstruction which responded to nonoperative management. There has been no other morbidity. The child who required the creation of a ventral hernia in order to return the viscera to the abdomen underwent anatomic closure of the abdominal wall at ten days of age.

DISCUSSION

Embryology: Diaphragmatic development is completed in the eighth week of gestation. The midgut normally returns to the peritoneal cavity from its extracoelomic position in the tenth week. If the bowel returns before the diaphragm is completed, or if there is failure of completion of the diaphragm, abdominal viscera will be displaced into the thoracic cavity. Lung development normally continues until the fourteenth week. Should viscera be displaced into the chest before the development of the lung is complete, some degree of pulmonary hypoplasia may result.

Distribution: Approximately 85% of diaphragmatic hernias are the result of posterolateral defects in the diaphragm, the foreman of Bochdalek. The left side predominates, being the involved side ten times more often than the left in infants who become

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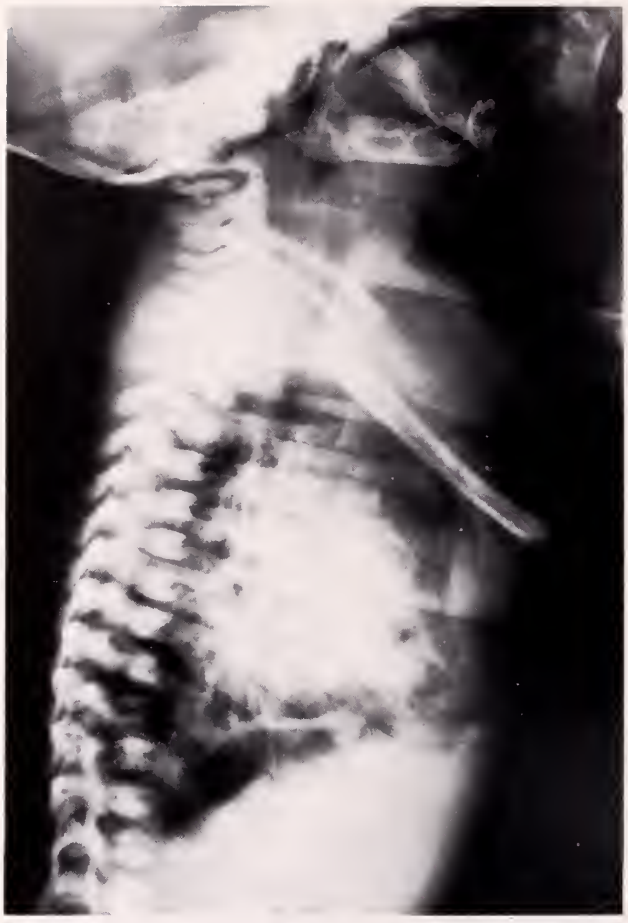

Fig. 1A

Fig. 1B

Fig. 1. Chest films showing gas filled viscera in the left hemithorax, with displacement of the mediastinum.

symptomatic within the first few days of life.[4] Male infants are affected twice as often as females.[4] As in our series, prematurity and other major anomalies are only occasionally noted.

Diagnosis: The diagnostic triad of dyspnea, cyanosis, and dextrocardia should alert the examining physician to the possibility of a diaphragmatic hernia. Decreased breath sounds, percussion dullness, and enlargement of the ipsilateral chest are often seen. Bowel sounds may be audible in the chest, and the abdomen will be flat due to lack of the normal amount of bowel in the peritoneal cavity. Confirmation of the diagnosis is readily obtained from plane films of the chest. The use of upper gastrointestinal studies is only rarely necessary. Generally noted radiographic findings are: (1) gas-filled viscera in the chest, (2) displacement of the mediastinum, (3) upward displacement of the liver in the right sided hernias, resembling an elevated right hemidiaphragm, and (4) mediastinal density in the anterior hernias of Morgagni.

Pathophysiology: The detrimental effects of the herniated viscera are several. First, there is compression of the ipsilateral lung, and with mediastinal shift, there is also compression of the contralateral lung. The mediastinal shift itself distorts the great vessels, and leads to decreased venous return to the heart. These mechanical distortions are in addition to the hypoplasia of the ipsilateral lung usually associated with this anomaly, and to the hypoplasia of the contralateral lung also noted in some studies.[5,6] The result of these derangements is hypoxia, with ensuing respiratory and metabolic acidosis, and rapid progression to the death of the infant.

Initial Treatment: Diaphragmatic hernia of the newborn demands urgent treatment. If this diagnosis is entertained, a nasogastric tube should be introduced immediately, in order to prevent more swallowed gas from entering the gastrointestinal tract and leading to increased volume of the bowel within the chest. The infant should be kept in a warm environment, his head should be elevated, and he should lie on the involved side. Oxygen may be given if indicated. If respiratory support is necessary, an endotracheal tube should be placed and

Fig. 2. The appearance at operation of the typical defect in the posterolateral aspect of the left hemidiaphragm.

Fig. 3. Autopsy examination revealing massive displacement of peritoneal viscera into the left hemithorax, with the hypoplastic left lung also demonstrated.

gentle positive pressure used. Bag and mask resuscitation is detrimental due to the large amounts of air often forced down the esophagus with this method. Determination of blood gases with appropriate sodium bicarbonate therapy may be indicated, and antibiotics are begun.

Operative Management: Arrangements for immediate operation should be made. These babies may suddenly change from an apparently satisfactory status to severe distress. The operating room should be warm, and a venous cutdown should be placed.

The abdominal approach is recommended for the usual left sided hernia. Advantages of this approach are the ease of repair of the defect while insuring protection of the abdominal viscera, and the ability to evaluate and repair the malrotation of the intestine associated with the hernia. We prefer a left paramedian, rectus retracting incision, although in urgent situations, a midline incision is preferable. Right sided posterolateral hernias are often best managed through a transthoracic incision through the ninth interspace.

The viscera are withdrawn from the chest, and the lung is examined. If a sac is present, it is excised, and the defect closed with interrupted, non-absorbable sutures. Posteriorly, the sutures must be placed superior to the upper pole of the kidney so as to prevent upward migration by the kidney, with potential upper urinary tract obstruction. In cases where insufficient diaphragm is available for primary closure, abdominal muscular flaps may be fashioned.[7] Before the last suture in the diaphragm is tied, a small catheter is passed into the chest and placed on suction. As the suture is tied, the catheter is withdrawn, evacuating as much air as possible from the chest, and creating a gentle negative pressure. No effort should be made to expand the hypoplastic lung, an excessive pressure on the small airways will lead to rupture and pneumothorax.

The intestinal tract is then examined for malrotation or other anomalies, and these are managed as indicated. A gastrostomy is created, and closure is accomplished. Occasionally, as in one patient in this series, it is not possible to return all of the viscera to the peritoneal cavity. Should this occur, only the skin and subcutaneous tissue is repaired, leaving a

ventral hernia which may be repaired in ten to fourteen days.

Postoperative Care: A chest film is obtained in the operating room, the baby is returned to the isolette, and again placed in the head up, ipsilateral side down position. Oxygen is given as needed, and blood gases are followed as indicated. Antibiotic therapy is continued for seven to ten days. Serial chest films will usually show return of the mediastinum to the normal position within a few days, with gradual expansion of the involved lung. Negative chest suction is usually not necessary. Feeding is begun when peristaltic activity returns, and the gastrostomy tube may be removed on the tenth to twelfth day.

During the postoperative period, the sudden recurrence of respiratory distress usually indicates pneumothorax, and this is treated with an intercostal catheter to water seal drainage.

Prognosis: Patients with congenital diaphragmatic hernia may be divided into two groups; a high risk group who develop symptoms within the first few days of life, and the remainder of patients whose onset of symptoms is delayed beyond this period. The latter group have essentially no mortality associated with the defect or its treatment. There is, however, a 40% to 50% mortality associated with diaphragmatic hernia in the high risk group.[3,4,8,9]

Eight of our patients fall into the high risk group, of whom two died, one before operation could be accomplished, and one shortly thereafter. Thus there was a mortality of 25% for the high risk group, which compares favorably with most reported series.

The ultimate prognosis for the survivors is excellent, with no evidence of impaired pulmonary function. Because of the excellent potential of immediate salvage, and the excellent long term outlook, it is most important that all physicians treating the newborn consider this diagnosis when evaluating an infant with respiratory distress, and should the diagnosis be established, arrangements should be made for urgent surgical attention.

ACKNOWLEDGMENT

The author wishes to thank Drs. Chris Lutes and Robert McAfee for their assistance in the preparation of this paper.

REFERENCES

1. Bock, H. B. and Zimmerman, J. H.: Study of Selected Congenital Anomalies in Pennsylvania, *Public Health Report* 82: 446, 1967.
2. Gravier, L., Dorman, G. W., and Votteler, T. P.: Congenital Diaphragmatic Hernia in Children, *Surg., Gyn., and Obs.* 132: 408, 1971.
3. Jackson, T. M.: Congenital Diaphragmatic Hernia, *Arch. Surg.*, 95: 102, 1967.
4. Johnson, D. G., Deaner, R. M., and Koop, C. E.: Diaphragmatic Hernia in Infancy: Factors Affecting the Mortality Rate, *Surgery* 62: 1082, 1967.
5. Campanale, R. P. and Powland, R. H.: Hypoplasia of the Lung Associated with Congenital Diaphragmatic Hernia, *Annals Surg.* 142: 176, 1956.
6. Kitagawa, M., Hislop, A., Boyden, E. A., and Reid, L.: Lung Hypoplasia in Congenital Diaphragmatic Hernia, *Brit. J. Surg.* 58: 342, 1971.
7. Simpson, J. S., and Gossage, J. D.: Use of Abdominal Wall Muscle Flap in Repair of Large Congenital Diaphragmatic Hernia, *J. Ped. Surg.* 6: 42, 1971.
8. Snyder, W. H., Jr., and Greavey, E. M.: Congenital Diaphragmatic Hernia: 77 Consecutive Cases, *Surgery* 57: 576, 1965.
9. Whittaker, L. D., Jr., Lynn, H. B., Dawson, B. and Chaves, E.: Hernias of the Foremen of Bochdalek in Children, *Mayo Cl. Proc.* 43: 580, 1968.

SURGICALLY CORRECTABLE HYPERTENSION, DIAGNOSIS AND TREATMENT

Continued from Page 200

Aortography should be performed in any patient in whom there is reasonable suspicion of renal artery disease and selective renal vein renin determinations should be obtained to determine the significance of the arteriographic findings.

Frequently other manifestations of the underlying arteriosclerotic process are present and must be investigated and dealt with simultaneously. Four cases of renovascular hypertension with a variety of associated problems are presented for illustration.

REFERENCES

1. Conn, J. W., Knopf, R. F., and Reed, M. N.: Clinical Characteristics of Primary Aldosteronism from an Analysis of 145 Cases. Am. J. Surg., 107: 159-172, 1964.
2. Poutasse, E. F. and Clifford, R. W., Jr.: Pheochromocytoma: Diagnosis and Treatment. Prog. in Cardiovasc. Dis., 8: 235-252, 1965.
3. Helmer, O. M.: The Renin-Angiotensin System and Its Relation to Hypertension. Prog. in Cardiovasc. Dis., 8: 117-128, 1965.
4. Morris, G. C., Jr., DeBakey, M. E., and Fanger, L. C.: Renovascular Hypertension. Surg. Clin. N. Amer., 46: 931-947, 1966.
5. Foster, J. H., Oates, J. A., Rhamy, R. K., Klatte, E. C., Burko, H. C., and Michelakis, A. M.: Hypertension and Fibromuscular Dysplasia of the Renal Arteries. Surgery, 65: 157-181, 1969.
6. Clifford, R. W., Jr. and Poutasse, E. F.: Renal Vascular Hypertension: Diagnosis and Treatment. Prog. in Cardiovasc. Dis. 8: 141-158.

Health Care and the 106th Maine Legislature

CHARLES L. CRAGIN III, Esquire*

At 12:20 a.m. July 4, 1973 the 184 members of Maine's 106th Legislature declared their collective independence from the legislative halls in Augusta and adjourned "without day," or, at least, for a while. The early morning adjournment brought to a close the longest legislative session in the history of the Maine Legislature. When it was over the Legislature had enacted 636 Public Laws (one of which the Governor vetoed); 75 Resolves; and 150 Private and Special Laws. Noting that the Legislature considered over 2000 legislative documents, a generalist would have to concede that it is easier to kill a bill than to pass one.

A look at the activities of this past session is useful for those in the health care industry for two reasons. Not only can we ascertain what changes have been brought about in the fields in which we deal on a daily basis but, also, we have an opportunity to examine that which did not become law. In doing so we may speculate as to which documents in the deceased category are harbingers of the future. This report is not intended to be an exhaustive analysis of any specific legislation but, rather, is intended to present a summary of the statutory changes in existing law, the enactment of new laws, and that legislation which was before its time.

The health care industry, composed of the various types of professional providers, hospitals, and third-party payors, had a busy session. Some energy was expended in "discussions" between the various professional groups; e.g. chiropractors versus physicians; optometrists versus opticians. Other energy was expended in a united effort by the entire industry in support of or opposition to specific legislation. Of the 636 bills which became public laws, for example, at least 52 had either a direct or derivative effect upon the health care delivery system in the State of Maine.

Early in the session, the Judiciary Committee took steps to resolve certain problems concerning the treatment of minors for drug abuse. Legislation, introduced by Representative Harold Silverman of Calais, was enacted permitting hospitals to admit minors without parental consent for a period of up to 16 hours in connection with the treatment of drug abuse problems. Such legislation was felt to be necessary because the 105th Legislature had granted physicians and other professionals the right to treat minors for drug abuse without parental consent. However, until now, hospitals had been required to secure parental consent.

Six days later that same committee watched the Legislature repeal the law making relatives financially responsible for persons receiving hospital care. The law, reportedly little used, was felt by the sponsor of the repealer, Representative Stephen Perkins of South Portland, to be unfair and "constitutionally suspect." On that same day the Governor signed into law an act requiring licensure of all facilities treating drug users unless such centers were otherwise licensed as medical care facilities. Similar legislation was enacted concerning community mental health facilities. Also signed into law was a bill permitting the Department of Health and Welfare to make Medicaid payments to hospitals on a more regular basis, thus helping to alleviate reported cash flow problems.

Municipal officials in York County were evidently perturbed with the handwriting of one or more physicians in the area. They convinced Representative Neil Rolde of York to introduce legislation requiring all death certificates to be typewritten. Expressing sympathy for the rural physician who was "overworked and not equipped with a typewriter," the Judiciary Committee amended the legislation to require that all death certificates be "typewritten or printed" prior to filing.

After unsuccessful attempts in the 105th Legislature, supporters of legislation controlling lead paint poisoning finally eked out a victory. The law, which controls the application of lead-based paint and places a duty on manufacturers and sellers to label containers, imposes an obligation upon physicians. Any physician knowing, or having reason to believe, that any patient has or is suspected of having lead poisoning, must immediately notify the Department of Health and Welfare. That Department may assist the physician in determining whether the case is one of lead poisoning and, if so, in locating the source of the poison.

The Licensure provisions dealing with osteopathic physicians were rewritten along the lines

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of the revised Board of Registration in Medicine law enacted in the 105th Legislature. So close was the parallel between the acts that the physician-patient privilege, inadvertently repealed by the 105th Legislature, was also not included in the new osteopathic law. The Legislature did, however, reinsert the qualified privilege into the allopathic law, and, beginning October 3, 1973, communications between an allopathic physician and his or her patient will again be privileged. Not so, however, for the osteopathic physician. Patients of Board certified psychiatrists, by other legislation, were given an unqualified privilege to refuse to permit a psychiatrist to divulge a communication.

On May 24, 1973, several months of legislative sparring between physicians and chiropractors came to an end — at least for this session. It had begun innocently enough, with the introduction of L.D. 1258, An Act to Improve the Quality of Health Care to all Persons. One of two legislative documents introduced by Senate President Kenneth P. MacLeod of Brewer, the bill simply proposed that chapter 19 of Title 32 be repealed. Shortly thereafter, it was discovered that chapter 19 of Title 32 licensed and regulated the practice of chiropractic! After substantial public debate, followed closely by the news media, the Legal Affairs Committee called for a public hearing on the issue. Senator Cyril Joly of Waterville, Chairman of the committee, did not know how many people would attend but was sure that it would be more than the small, fourth floor committee room could accommodate. He took no chances and reserved the capitol city's new Augusta Civic Center. He was right! Conservative estimates placed the crowd at over 800, and, when Senator Joly called for a show of hands, it appeared to be 650 pro-chiropractor and 150 against. Subsequent to the 6-hour hearing, the Legal Affairs Committee apparently decided that doing away with chiropractors might not be the most politic thing to do. They did, however, recognize that certain changes in the chiropractic licensure law were necessary. On May 24th, Governor Kenneth Curtis signed into law a bill requiring that all chiropractic candidates for licensure be graduated from a nationally accredited chiropractic college. The bill also prohibited advertising and set forth standards for telephone listings and business cards. It prohibited chiropractors from using informational lettering on doors, signs, and windows, and from placing announcements in newspapers, etc., except as such action was "deemed ethical by other health practices of the community." The legislation also set forth the various grounds upon which a license to practice chiropractic could be revoked.

"Due Process by Governmental Agencies" was the theme of legislation requiring all governmental agencies, including the various professional licensure boards, to keep a written record of every decision involving the approval or denial of a license. This record must be available for public inspection. If the agency denies a request for licensure, it must set forth the reasons for such denial.

Two bills dealing with physician's assistants and nurses in an extended role were enacted. These bills were responses to a Legislative Study Committee's recommendation that would have placed great control over physician's assistants with the Department of Health and Welfare. The Committee's bill would also have set up a stringent licensure system for such assistants. Arguing that now was not the time to set up another intricate and unwieldy system of licensure, especially in such a new and developing field, health care professionals joined forces and pressed hard for enactment of "delegatory" statutes. Under these laws a registered nurse may diagnose illness or prescribe therapeutic or corrective measures when he or she has such duties delegated by a physician. Prior to legally performing such duties, the nurse must have completed the necessary additional educational program approved by the Board of Nursing and the Boards of Registration in Medicine and Osteopathic Examination and Registration. However, for some unexplained reason, this legislation will not become effective until October of 1974.

The law specifically dealing with "physician's assistants" permits allopathic and osteopathic physicians to delegate to an assistant any medical activities or services, so long as the assistant has completed a training program approved by the applicable board of registration, and the assistant is under the control and supervision of the physician. The law does not, however, prohibit a physician from "delegating to his employees certain activities relating to medical care and treatment now being carried out by custom and usage when such activities are under the control of and in the personal presence of the physician or surgeon." Lest someone think this is the quick answer to the workload problem, one caveat is appropriate. The law goes on to say: "The physician delegating such activities . . . shall be deemed *legally liable* for such activities of such persons. . . ." (Emphasis added). Since the Physician's Assistant law goes into effect on October 3, 1973, and the modification in the Nurse Practice Act is not effective until October 1, 1974, it would appear that any registered nurse operating in an extended role prior to the latter date will be in violation of the Nurse Practice Act. This conclusion is apodictic since the Physician's Assistant law specifically excludes registered nurses from its operation.

Another way to increase the supply of blood donors was suggested by Representative Domenico Santoro, a Portland physician. Dr. Santoro pro-

posed that prisoners in state institutions be given a sentence reduction of five days for each pint of blood donated to various specified sources. Under this bill, signed by the Governor on June 13th, each prisoner would be permitted to donate one pint of blood each six months. Subsequent to its enactment, however, the American Red Cross and the U. S. Department of Health, Education and Welfare publicly stated that such donations were not to be encouraged since they would constitute donations for compensation. Evidently, those agencies are concerned about the falsification of medical records which might occur in order to secure the reduction in sentence.

A highly emotional issue, precipitated by companion cases announced by the United States Supreme Court in February, lingered in the legislative process until the middle of June. The gestation period having been concluded, the Judiciary Committee, chaired by Senator Wakine Tanous of Millinocket, reported out two bills dealing with abortions. The comparatively non-controversial document, originally introduced by Representative Louis Jalbert of Lewiston, granted immunity to physicians, nurses, and other persons who refused to perform or assist in the performance of abortions. The legislation also prohibited discrimination in employment or admission due to the refusal of a person to assist in an abortion; and it placed a criminal penalty upon any person using or transferring a "live human fetus, whether intrauterine or extrauterine . . . for scientific experimentation or any form of experimentation." The legislation did, however, inject certain problems for the physician performing an abortion. The bill defined "live birth" as

> "a product of conception after complete expulsion or extraction from its mother, irrespective of the duration of pregnancy, which breathes or shows any other evidence of life such as beating of the heart, pulsation of the umbilical cord or definite movement of voluntary muscles, whether or not the umbilical cord has been cut or the placenta is attached."

Whether such a definition causes problems to the physician performing an abortion will, I assume, depend upon the medical procedure utilized. However, the physician should be forewarned that the legislation also says that when such a "live birth" occurs,

> "failure to take all reasonable steps, in keeping with good medical practice, to preserve the life and health of the live born person shall subject the responsible party or parties to Maine law governing homicide, manslaughter and civil liability for wrongful death and medical malpractice."

After some parliamentary maneuvering, the bill was enacted and approved by the Governor. It will become law on October 3, 1973.

The controversial bill dealing with the same subject area was introduced by Representative David Huber of Falmouth. Huber, responding to the U. S. Supreme Court decisions, sought a law that would require second and third trimester abortions to be performed in a hospital and that would prohibit abortions in the third trimester except to save the life or health of the mother. Members of the Maine Right to Life Committee opposed this legislation arguing that enactment would affirm the decision of the Supreme Court. The legislation was defeated, and Maine is now left with no law controlling the locations where abortions may be performed or the circumstances under which they may be performed in the third trimester. It also leaves Maine law devoid of any legislative statement as to whether the consent of the father or husband is required prior to the performance of an abortion.

Crisis developed late in the session with the public announcement of an opinion of an Assistant Attorney General that personnel not licensed as nurses were "administering" medications at state institutions and that such was in violation of law. Those personnel concerned immediately ceased such activities for fear of criminal penalties, and the medication delivery system at those institutions came to an immediate standstill. Representatives of the various health professions met in Augusta and determined that it was impossible to conduct a review of the various licensure laws in order to propose revisions before the Legislature adjourned. Therefore, it was recommended that legislation be enacted to preserve the status quo until such time as the area of administration of medications could be comprehensively reviewed by the health care professions. Representative Domenico Santoro of Portland consented to the utilization of a legislative document which he had sponsored as the vehicle, and it was totally amended to grant immunity to non-licensed personnel performing "selected activities in the administration of medications" until July 1, 1974. Enacted as an emergency, the law became effective on June 20, 1973.

Late in the session the Legislature also enacted bills which totally revised the laws governing admission to mental health facilities and which created the Uniform Alcoholism and Intoxication Treatment Act. During the waning hours, legislation requiring the immediate reporting of certain information concerning the Sudden Infant Death Syndrome was enacted along with legislation ordering the Department of Health and Welfare to establish family planning programs. Interestingly enough, the Family Planning law allows family planning services to be rendered to minors who are parents, married, or "who may suffer, in the professional judgment of a physician, probable health hazards if such services are not provided."

The Legislature, in its final days, also made substantial commitments of money to the health care professions by granting funds to regional planning agencies; Medical Care Development, Inc.; allopathic and osteopathic scholarships; funds for the treatment of cystic fibrosis; and $100,000 to begin work on a medical school for Maine.

This, then, is a brief and, albeit, rather sketchy synopsis of some of the affirmative actions taken by the Legislature in the health care field. At the same time, the Legislature turned thumbs down on legislation designed to make hospital "incident reports" privileged information and thus immune from discovery by interested trial attorneys. It reviewed with some interest a proposal by Senator Gerard P. Conley of Portland to establish a state health insurance program for every Maine resident. While the bill in its original form was impractical due to the high cost ($325 million per year), the Legislature did establish a special study commission to investigate the subject matter and to report to the next Legislature. The Governor is required to appoint representatives from the Maine Medical Association, Maine Hospital Association and Maine Blue Cross and Blue Shield to serve on this commission.

The Legislature declined to order third-party payors to pay psychologists if they paid psychiatrists; or optometrists if they paid opthamologists; or chiropractors if they performed any services. It also stopped short of ordering insurers and Maine Blue Cross and Blue Shield to pay the same benefits for mental illness as is paid for physical illness. Legislation requiring all insurers and Blue Cross to provide the same maternity benefits to unmarried women as is provided to married women was enacted in the House but died on the Special Appropriation Table on the last day of the session.

Legislation placing controls on the establishment of Health Maintenance Organizations and the Licensing of Opticians died late in the session, but both will probably be studied by Legislative Committees.

A bill proposing that pharmacists be permitted to dispense generic drugs even though the physician had specified a "name brand" drug came close to passage. Not as close, however, as two bills permitting druggists to advertise the price of drugs.

The list is seemingly endless. Out of over 2000 bills dealt with by the Legislature, at least 10% dealt with some aspect of the health care industry, including legislation designed to remove the property tax exemption from hospitals.

What message can be gleaned from this? Simply stated, it is that the health care delivery system is being subjected to public and legislative scrutiny in a dimension that it has not experienced until now. Health care providers must, of necessity, take positive steps to educate themselves concerning the legislative process and must take similar steps to educate their legislators as to the complexity of the health delivery system. Legislators desire, value and respect the input of knowledgeable persons and groups who stand ready, willing and able to assist them and to provide information. As one veteran legislator opined recently: "If they don't help us when we build the wheel, they'll certainly be up here later trying to fix it." And, just in case the Legislature didn't happen to take action on your pet concern, take heart, THEY'LL BE BACK NEXT JANUARY (or maybe earlier)!

Fall Meeting of the M.M.A. House of Delegates

Sunday, December 9, 1973

St. Joseph Hospital, Bangor, Maine

12:30 P.M. — Registration; 1:00 P.M. — Lunch; 2:00 P.M. — Meeting

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10:00 A.M. — Meeting of the Executive Committee

THE PHYSICIAN AND BLUE SHIELD — A NATIONAL PERSPECTIVE

The following is part of a speech presented by the outgoing Chairman of the National Association of Blue Shield Plans (NABSP) Board and speaks to the unique features of the physician-Blue Shield agreement.

"The real leaders of the health care industry are those who cause the right things to happen," retiring NABSP Board Chairman Ira C. Layton, M.D., told the Blue Shield Annual Meeting Delegates.

"In the three years I have been privileged to chair this Board, and in the previous four years when I served as vice chairman," observed Dr. Layton, "I believe there have been more changes in the approach to health care than occurred in many a decade of previous eras."

Enumerating these innovations, the Kansas City internist cited the Usual, Customary and Reasonable charge program, increased emphasis on diagnostic and ambulatory care coverage, the development and implementation of the Blue Shield Reciprocity program, and the continued growth of the Blue Cross and Blue Shield Federal Employees Program. He also made mention of the use of computers in medicine and EDP technology in claims processing utilization review and data retrieval; the rise of consumerism in health care delivery and financing; development of alternate delivery systems; the emergence of medicine's role in assuring quality care; and the possibility of some form of National Health Insurance being enacted.

Discussing consumerism, he noted that millions of people have now been educated to think they are entitled to a respected and productive role in society.

"An informed public paying high prices whether through taxes, dues, bargaining or cash for a product they feel they must buy, whether meat or health care, will immediately demand a voice in determining the specifications," he contended.

Physician Participation

Moving to the subject of physician involvement in Blue Shield activities, Dr. Layton told the group, "Blue Shield is the only mechanism — the only arena — in which the physician and the third party representative can sit down together in an official capacity.

"To the extent that these efforts are successful," he hypothesized, "Blue Shield has become the largest voluntary effort by medicine to serve Americans."

Dr. Layton emphasized that the contributions of many physicians to Blue Shield go far beyond mere criticism. He specifically cited the countless hours physicians have given free of charge on peer review committees and boards of directors.

"This expertise and labor have helped Plans construct sophisticated utilization review programs which encourage the efficient delivery of quality medical service at the lowest possible cost," he observed.

In summary, Dr. Layton stated: "The sense of security many of us had back in the 1950's belongs to that more comfortable age. There has always been an awareness at Blue Shield that the problems and the challenges facing us are great, but that the opportunity and the rewards are greater if we work at getting optimum results."

Available from Maine Blue Shield

A chart listing all the current National Health Insurance bills in Congress with a brief description of each bill is available by writing:

Public Relations
Maine Blue Cross and Blue Shield
110 Free Street
Portland, Maine 04101

Necrologies

Clement S. Dwyer, M.D.

1915-1973

Dr. Clement S. Dwyer, 57, of Bangor, Maine died on June 5 in a Bangor hospital.

He was born in Boston, Massachusetts on October 18, 1915, son of William J. and Marion W. Dwyer.

Dr. Dwyer was graduated from Harvard University in 1937 and received his medical degree from Tufts University School of Medicine in 1941. He interned and served a residency in anesthesiology at the Rhode Island Hospital in Providence. From 1941 to 1946, Dr. Dwyer served in the U. S. Army Medical Corps with the 128th and 133rd General Hospitals in Leyte, Philippines. He was chief anesthesiologist at the Eastern Maine Medical Center from 1947 to 1971, retiring in May 1972.

He was an affiliate member of the Penobscot County Medical Society and the Maine Medical Association. Dr. Dwyer was also a member of the American Medical Association, the American Board of Anesthesiologists, the American College of Anesthesiologists, the International Anesthesia Research Society, the American Society of Anesthesiologists, the New England Society of Anesthesiologists and the Maine Society of Anesthesiologists.

Dr. Dwyer had served as a past officer of the Tufts Medical School as the Charlton Research Fellow in physiology from 1941-to 1942, the Maine Society of Anesthesiologists, the medical staff of Eastern Maine Medical Center, the Bangor Medical Club, the Penobscot County Medical Society and the New England Society of Anesthesiologists.

Surviving are his widow, the former Marguerite L. McKay M.D. of Bangor; two sons, Clement S. Dwyer, Jr. of Brooklyn, New York and Thomas M. Dwyer of Bangor; one brother, William J. Dwyer of Woburn, Massachusetts; and his mother of Wakefield, Massachusetts.

Harry Butler, M.D.

1898-1973

Dr. Harry Butler, 74, of Bangor, Maine died unexpectedly July 4 at his residence.

He was born in Bangor on July 12, 1898, son of Harry and Caroline N. Butler. His father was a physician, also.

Dr. Butler was graduated from the University of Maine in 1920 and received his medical degree from the University of Vermont College of Medicine in 1925. He served in the Army in World War I and in the Air Corps during World War II. He practiced in Newmarket, New Hampshire and then located in Bangor in 1928, where he specialized in Otolaryngology.

He was a senior member of the Penobscot County Medical Society, the Maine Medical Association, the American Medical Association and the American Board of Otolaryngology.

Surviving are his widow, the former Irene Doran of Bangor; two daughters, Mrs. David Dean of Buffalo, New York and Mrs. George May of West Newton, Massachusetts; one brother, Theodore Butler of Grafton, Massachusetts; six grandchildren and one nephew.

Arthur B. Woodman, M.D.

1902-1973

Dr. Arthur B. Woodman, 70, formerly of Falmouth, Maine and a resident of Spring Hill, Florida since his retirement in 1971, died on July 23 at a local hospital.

He was born in Yarmouth on October 27, 1902, son of Daniel and Hattie K. Woodman.

Dr. Woodman was graduated from Bowdoin College and received his medical degree from Boston University School of Medicine in 1936. He practiced in Vinalhaven, Maine before joining his physician-father in practice in North Haven, Maine. In 1939, Dr. Woodman located in Falmouth.

He was a senior member of the Cumberland County Medical Association, the Maine Medical Association and the American Medical Association. In 1969, the Falmouth Lions Club named Dr. Woodman "Falmouth Citizen of the Year."

His first wife, the former Charlotte Gibson, died in 1970.

Surviving are his widow, the former Helen Moyer; a son, Lieutenant Commander Daniel Woodman with the U. S. Navy at Fort Deitrich, Maryland; a daughter, Mrs. Janice Alden of Sudbury, Massachusetts; three sisters, Mrs. Ruth Verrill of Gray, Maine, Mrs. Sarah Brown of Freeport, Maine and Mrs. Alice Grant of North Haven, Maine; and three grandchildren.

News, Notes and Announcements

American Cancer Society's
National Conference on Childhood Cancer
May 16-18, 1974
Fairmont Hotel
Dallas, Texas

The purpose of this conference is to alert the medical community to the progress being made in the diagnosis and treatment of childhood cancer and to emphasize the need for early diagnosis and the importance of prompt treatment by a team of medical specialists and allied health personnel capable of rendering optimal care.

Sessions are open to all members of the medical and related health professions. Pre-registration is requested. There is no registration fee.

This program is acceptable for:

- 15 Credit Hours in Category I for the Physician's Recognition Award of the American Medical Association
- 15 Elective Hours by the American Academy of Family Physicians

For information write: American Cancer Society's National Conference on Childhood Cancer, 219 East 42nd Street, New York, New York 10017.

International Seminar
on
Cardiovascular Disease

Symposia Medica Foundation, in cooperation with The Royal Society of Medicine, presents an international seminar on CARDIOVASCULAR DISEASE to be held in London, October 12-20, 1973.

Chairman: Professor John P. Shillingford, President, British Cardiac Society

Registration: $100.00

Further information, contact: Cynthia Soika, M.A., Projects Director, Symposia Medica Foundation, 305 East 24th Street, Suite 17-F, New York, N.Y. 10010.

County Society Notes

PENOBSCOT

The April meeting of the Penobscot County Medical Society was held on April 17, 1973 at the Tarratine Club in Bangor, Maine.

The minutes of the March 1973 meeting were read and approved.

A letter from Governor Kenneth Curtis in reply to the resolution on chiropractic submitted by this County Society was read. A communication from a resident of Old Town, Maine, requesting the Society's help and aid in the recruitment of a new physician for the Old Town area was noted. A letter from the University of Maine, Conferences and Institutes Division, advising the Society that the facilities of the University of Maine will be available during this summer period for conferences and sessions was read.

Dr. Thornton W. Merriam, Jr. reported that as a result of a recent meeting of the Executive Committee of the Maine Medical Association, an assistant to the Executive Director, Dr. Dan Hanley, would be appointed. It is assumed that part of his responsibility would be to work with and keep informed the County Societies in regard to Health Care Organizations and Delivery Systems. This, in large part, answered the question raised by the membership of this County Society at the February 1973 meeting concerning the need for an individual or individuals keeping the members of the Society informed in the field of Health Care Delivery Systems.

A report of the House of Delegates meeting held on April 8, 1973 at Thayer Hospital, Waterville, Maine was read.

Dr. George W. Wood, III provided a report concerning the status of the PSRO at the State level and ultimately at the county level. He reiterated the necessity for we as a Society to think in terms of PSRO and to develop criteria of care together with length of stay data so that it may be applied by ourselves at our own local level.

A motion was made, seconded, and passed to instruct the delegates of the Society to the House of Delegates to vote in favor of a resolution concerning continuing education to be presented to the State Society at its annual meeting in June 1973, which states that each member be required to report to the State Society his activities in the field of medical education each January 1 of each year.

Dr. Dexter J. Clough, 2nd, the President-elect, appointed a nominating committee consisting of Drs. Shapero, Houlihan and Wood, to submit a slate of officers for the coming year. This report will be submitted at the annual meeting of this County Society in May 1973.

The speaker for the evening, Dr. Robert Graves, from the University of Maine Health Services in Orono discussed his desire for the formation of a Family Health Center at the University of Maine in Orono. He desired that the greater Bangor medical community participate in this program, by providing care to enrollees of this plan. As described by him, the Family Health Center would be a prepaid medical program for primarily the faculty and staff of the University of Maine at Orono and would have several physicians employed by the plan based at the University. Medical support and specialized care and facilities would then hopefully be provided by physicians of the greater Bangor area. He asks for the support of the Society in this endeavor.

There being no further business, the meeting was adjourned.

PHILIP G. HUNTER, M.D., *Secretary*

YORK

The May meeting of the York County Medical Society was held on Wednesday, May 9, 1973 at the Webber Hospital, Biddeford, Maine. It was an evening meeting that started with a social hour from 6:30 p.m. to 7:30 p.m. with dinner and business meeting following.

The featured speaker was Dr. George W. Wood, III, of Brewer, Maine, who is President of the Maine Medical Association. His subject was, "The Maine Medical Association Action Program." He discussed many facets relative to this subject and gave a very illuminating talk.

Dr. Carl E. Richards, President of the York County Medical Society, gave a revealing talk on the recent Chiropractic meeting in Augusta which he attended. He is a tremendous individual and certainly did a masterful job, but after all he is only one individual. He is a tireless worker, and in my estimation has done much to further the cause of Medicine in the State of Maine. I frankly don't know how he does it all.

It is interesting to mention at this time that two camperships

for Diabetic Children, ages 8 to 14, to attend the Salvation Army Summer Camp at Lake Sebago have already been selected due to the efforts of Drs. Ross, Moore and Page of Saco. Good work! This was reviewed briefly by Dr. Lyman Page.

The financial position of the York County Medical Society was reviewed and I was happy to report that we are quite well off financially.

The following Committees were appointed by the President, Dr. Carl E. Richards. They are as follows: Public Health — Drs. Lyman A. Page, Alexander W. Magocsi and Leandre R. Charest; Medical Disaster — Drs. James S. Johnston, Alvin A. Hoffman and Marion K. Moulton; Medical Recruitment — Drs. George A. Lord, Marcel P. Houle and Herman G. Sandvoss; Care of the Aged — Drs. Harry Lapirow, Melvin Bacon and S. Dunton Drummond; Auditing — Drs. Melvin Bacon and Lawrence R. Hazzard; Resolutions — Drs. Robert D. Vachon, Andre P. Fortier and Conner M. Moore; and State Committee on Drug Abuse of Lawyers and Physicians. York County Medical Society Appointee — Dr. Maurice Ross.

Dr. Richards indicated that the next York County Medical Society meeting would be held at the York Hospital, York, Maine on October 10, 1973. It will be an evening meeting. The committee in charge of this meeting is composed of Drs. Kenneth E. Leigh and Lawrence R. Hazzard.

It was also mentioned that new application blanks for the County Society members will be forthcoming soon.

Four possible sites were selected as potential places for the Annual Meeting of the York County Medical Society and its Auxiliary to be held on January 9, 1974. After due consideration, it was decided to hold this meeting at the Officers Club, U.S. Navy Yard, Kittery, Maine. It can be stated here that we will have a speaker of national repute and that there will be several well-known national figures attending. We promise you an exciting evening.

It was also mentioned that the "Workshop on Stroke" for the nurses of Southern Maine, which was held at Nasson College in April, went over with a bang. There were about 200 R.N.'s, L.P.N.'s and student nurses attending.

A letter from Charles Came, who is interested in the Family Nursing Associate Program, was discussed. He has a tremendous background and if any physician is interested in sponsoring him, please let your secretary know.

It was also announced that the Annual Meeting of the Maine Medical Association is to be held at the Shawmut Inn, Kennebunkport, Maine, June 16 to 19, 1973.

The meeting was attended by 24 physicians and guests, which is much better than the average attendance. The meeting was adjourned at 10:15 p.m. with everything in complete accord.

MELVIN BACON, M.D., *Secretary*

CUMBERLAND

The 378th meeting of the Cumberland County Medical Society was held on May 17, 1973 at Valle's Restaurant. Seventy-two members were in attendance. Since this was our annual meeting, there was no special program. Following a brief social hour, an excellent dinner was served. It is the general opinion of the group that the quality of meals and service here has been much better than average throughout the year.

Dr. Douglas R. Hill called the business meeting to order at 8:00 p.m. Reading of the minutes of the last meeting was omitted following the passage of a motion to permit this.

A letter was received from Dr. Philip J. Keating announcing his relocation in Massachusetts and his resignation from Cumberland County Medical Society. A second reading of the application of Dr. Donald E. Bittermann was made and Dr. Bittermann was unanimously elected to junior membership in the Society.

First reading of the following applications were made. These are all for junior membership: Drs. Donald Leeber, Joseph Markee, Joseph Wilkis and Charles Thurber. The applications will be forwarded to the Executive Committee and presented to the membership for final vote in September. Dr. Hill announced that the Executive Committee decided against supporting financially the medical students coming to Portland under the SAMA Program. He further announced that the CCMS telephone answering service is now functioning. Our second television show will be aired on June 8 at 8:00 p.m. over Channel 13. The stars of this month's presentation will be Drs. Thorpe, Hiebert, Carroll and Hill and in an outstanding role, the Gray Ambulance Unit.

The following annual committee reports were presented: Professional Ethics — Dr. William L. MacVane, Jr.; Auxiliary Committee on Legislation — Dr. Charles W. Capron; Utilization Review — Dr. David S. Wyman; Health Insurance — Dr. Ferris S. Ray; Bylaws — Dr. Sidney R. Branson; Medical Legal Liaison — Dr. Stephen E. Monaghan; Medical Legal Screening — Dr. Stanley B. Sylvester; Health Careers — Dr. Roland G. Ware, Jr.; Peer Review — Dr. Harry A. Bliss; and Economic Affairs — Dr. Robert G. Sommer.

The treasurer's report was then presented and shows a balance of $5,688.74 in the checking account, with $1,800 in the savings account.

Dr. Hill presented the annual President's report and included an excellent summary of the past year's activities.

Following this, four resolutions which will be introduced at the annual meeting of the House of Delegates of the Maine Medical Association were presented. The first is a resolution originating from Dr. Richard T. Chamberlin in Waterville, Chairman of the Continuing Education Committee. This resolution may be seen in its entirety in the agenda for the upcoming meeting. The Cumberland County Medical Society voted to endorse this concept.

The second resolution originating from Penobscot County concerning the Chiropractic issue was tabled. A third resolution originating with Dr. Benoit Ouellette suggesting non-compliance with HR 1 and PSRO was also tabled.

Finally, a fourth resolution originating from the Cumberland County Medical Society was presented by Drs. Ronald J. Carroll and Robert E. McAfee. This was unanimously passed.

Following this, the Nominating Committee presented its report. The following delegates were appointed:

One Year Delegates: Drs. John R. Davy, Irving J. Poliner, Peter B. Webber, Robert G. Sommer, Stephen E. Monaghan and Douglas R. Hill.

One Year Alternates: Drs. Harry A. Bliss, William J. Hall, III, Wesley J. English, Hugh P. Robinson, Paul Maier and Kirk K. Barnes.

Two Year Delegates: Drs. Ronald J. Carroll, George I. Geer, Jr., John F. Gibbons, Walter B. Goldfarb, William L. MacVane, Jr. and Donald F. Marshall.

Two Year Alternates: Drs. Newell A. Augur, Jr., Elio Baldini, Carl A. Brinkman, John T. Dinan, Jr., Robert H. Pawle and Roland G. Ware, Jr.

The above appointments become effective following the annual meeting of the Maine Medical Association in June. The present appointees will continue as delegates and alternates until that time.

Dr. Lawrence Crane, Chairman of the Nominating Committee, then presented nominations for the offices of President, Vice-President, a member of the Professional Ethics Committee and a member of the Executive Committee.

Dr. Douglas R. Hill was re-elected to a second year as President of the Society. Dr. Winton Briggs was elected to a second year as Vice-President of the Society. Dr. Morrill Shapiro was elected to a five-year term as a member of the Professional Ethics Committee; his appointment to expire in 1978. Dr. Harry A. Bliss was elected to a three-year post on the Executive Committee; his appointment to expire in 1976.

There being no further business to come before the Society, the meeting was adjourned at 10:00 p.m.

ALFRED E. SWETT, M.D., *Secretary*

The Journal
of the
Maine Medical Association

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Quo Vadis, Medicus?

STEPHEN B. DEWING, M.D.

(The writer is obviously suffering from a sense of alienation, or future shock. He grew up with an idealized 19th Century image of medicine and doctors, and has never quite gotten over it. He believes, for instance, that good manners and compassion are basic equipment, that medical art is just as important as science, and frequently more so.) – SBD

Many medical persons at this point in time see themselves in a rather vulnerable and defensive posture. The individual doctor may feel secure in his grasp of the tools of his trade, his honesty in respecting his own limitations, the affectionate regard of most patients, but — he feels very insecure about his ability to control his future, particularly the financial and administrative aspects (national health insurance), and even more his professional status.

The question of status derives from the concern which has been rising rapidly in recent years both inside and (especially) outside medicine over the need to maintain professional competence in a field which has obviously been changing and developing with breathtaking speed.

Everyone agrees that continuing education is needed. Indeed, it is built-in to medical tradition that a doctor continues to learn from experience and from mistakes, and thereby eventually achieves a degree of judgment and maturity which the new graduate wants. But that sort of learning is no longer enough.

The problem is: how to ensure and measure the continuing competence demanded by the non-medical community.

Continuing education is currently in a rather chaotic state. Every hospital or clinic is now a "medical center"; everyone and his uncle is rushing to get into the act. Courses in everything, and in all combinations, are available and announcements reach every doctor's desk in handfuls (I have been receiving several per week routinely, and sometimes as many as 6 or 7 at a time — just within my own specialty of radiology). There is supposed to be some magic in racking up so and so many hours of courses, and some medical societies and hospital staffs are supporting this magic by *requiring* certain hours of instruction per year. These programs are (so far) all voluntary, but the most important point is that there is no guarantee that the trainee learns or applies anything, nor is there any way to measure results. The present usual format of most 2-5 day refresher courses is open to serious criticism. Even though a program is well designed and the faculty works hard, there is much to suggest that very little of important lasting usefulness is learned by a large number of students. Furthermore, quite a few doctors are extremely busy with their own practices, or cannot easily obtain coverage, so they simply do not attend any courses at all.

Personally, I think that continuing education of most specialists can probably be conducted successfully through their specialty societies — if membership is compulsory, and attendance at certain educational programs is also required. For the general practitioner I believe that continuing education (to be effective) must, in general, be brought to him in a form which is attractive, rewarding and compulsory. Here may be a rejuvenating role for the State and County Medical Societies.

The other major idea for enforcing medical competence is periodic re-examination, re-licensure or re-certification. This would be a new

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Bargaining: A System of Marital Counseling for the Family Practitioner

PHILIP C. GROCE, M.D.

Every practitioner of medicine finds at times that he has to do marital counseling. Husbands or wives present with physical complaints which may turn out to be non-organic. Once the psychological circumstances are explored, the physician finds a marriage on the rocks, or close to it. What to do then, is the problem. Will the physician refer the couple for psychiatric treatment, knowing the expense involved? Will he give the patient tranquilizers? Will he see the couple himself in therapy sessions knowing that he has very little time to do on-going psychotherapy in his office?

Once any marital relationship has reached the point where willing compromise is not made, tensions and friction mount. The marital partners try old patterns of adapting and when they fail, they usually seek help outside the family. At that time, the couple needs the introduction of a (new) system for resolution of the crisis. The physician (or some other care giver) must introduce that system.

As a busy practitioner of medicine, I have had to develop a system which would be psychologically sound but put most of the work on the couple themselves, i.e., it had to be basically a "self-help" system. I have developed a system which I call *Bargaining*. Bargaining is based on the behavioristic school of psychology. If acceptable to the couple, the system needs little supervision. The primary role of the physician in the system is to screen out physical disease and psychosis and to be available as a sympathetic witness as the process proceeds.

I will describe bargaining as if addressing the couple seeking help. The explanation should be given to the patients in the form of a pamphlet as well as verbally.

Bargaining

You find yourself in a relationship with your mate. When you first met, you certainly felt something that seemed like love. But since then, things have degenerated considerably. You are not too sure you still love him (or her); you argue a lot; and you complicate your life in so many ways because it is almost a blessing not to be around him (or her) very much alone. At days end you fall into bed exhausted, benumbed, and turned off. If you have had the experience of being married two, three, or even four times you learn that it is the same pattern over and over again. The grass really is not greener; it only seems that way.

If you want to break the dull stalemate in your present relationship and you want to resolve the recurring arguments that cause tension between you and your mate, then you want to develop a *growing relationship*. To do so, you have to be willing to expend some time and effort. You will have to set aside time to be together. That means putting the relationship ahead of your work — for the time being.

If you are dissatisfied with your present mate but you do not want to modify your life in any way, then you can pretty much plan that the relationship will end sooner or later. And, for some relationships, perhaps, termination is the best solution — especially those relationships where one of the partners is solidly addicted to alcohol or other drugs. Bargaining however is for people who wish to take the time and make the effort on the chance that their relationship can be improved.

If you wish to use the system of bargaining, you must adhere to four rules — the four commandments of bargaining. The rules state a necessary set of circumstances to change a stalled relationship into one that is growing.

Rule I: Rule one states that if there is a problem that produces friction, the problem resides in both partners 50:50. It is the problems that are in common with two people that produce friction. Another way to put it: it is the neuroses we have *in common* with significant people in our lives that cause the friction. We are all neurotic (and that is ok!), and much of that which is neurotic is accepted by people with whom we have close relationships. That which is *not* accepted and causes friction, is that problem or neurosis which is in common with both people in the relationship. So each problem has a "flipside" in the mate.

Rule number one has to be accepted on faith, because at the very beginning of the growth process, the 50:50 character of the difficulties is never apparent to either party (one partner usually appears to be a "victim"). Nor can this equal sharing of the problem be adequately explained so that it will be totally accepted by either party.

It is important to realize that the friction produced by problems within each partner generally ends up focussing on sex. The sexual aspects of the relationship are nearly always affected. As a practitioner of medicine, I frequently see people asking for help in what they think are primarily sexual problems. But in my experience, I have found that very rarely, except in the young, is the problem just sexual. Sexual difficulties that have

arisen fairly acutely are nearly always symptoms of other underlying friction between the partners.

Rule II: The second rule states that if you have been in a relationship for a relatively long time with your mate, assume for the time being, at least, that you are in love. You may feel numb, angry, jealous, depressed, and you may doubt your love; but assume that you are in love, anyway. And most importantly, assume that you feel toward him or her as much love as you could feel toward anyone at this time. It is possible, with careful unravelling of a problem, to attain that state of good feeling you had at the beginning of your relationship. Love is a gift, a gift which you give *to* someone. Once given, it cannot be taken away — except by doubt. Doubt is the big enemy of love. As soon as the love is doubted, it goes away. Or rather, the feeling goes away.

Rule III: Rule number three is to discard "grass is always greener" fantasies. It is fantasy to say, "I would be happy if only my mate were more intelligent (or less intelligent), were more beautiful (or less beautiful), had smaller hips and a larger bust (or visa versa), etc., etc. Each person has his quota of "good" qualities and unfortunate qualities as far as you are concerned. And if you think that it will help to change partners in order to find someone that has more "good" qualities, then you are indulging in fantasy. Realizing the perfect partner does not exist, you need to be prepared to pay the price for the qualities in your partner that you like. The price you pay is having to accept the qualities that you do not like.

Rule IV: Rule number four is that both partners will have to change equally. There will be too much resentment (overt or covert) if one changes and the other does not.

If you are inspired enough to accept the ground rules, then you are ready to work and get the relationship moving. Bargaining is simple in concept; yet, it has sound psychological principles behind it. It will work if you have faith in it and you do not get too discouraged when the going gets rough. The very least this method can do is get you and your mate together to talk about your problems. The very most it can do is solve the problems. Bargaining gives you a framework for change. It is a system.

Here is how it works. You and your mate will have to look at each of the situations that produce the recurrent friction between you. Each of you will have differing opinions as to the cause. Cause does not matter. On a piece of paper, write down what each thinks the *other* can *do* to alleviate the friction. Each of you will end up with a list of things that you think your mate should do to reduce the tensions. The lists can be drawn up together or in private — it does not matter.

Now the bargaining begins. You will trade items or groups of items listed. "I will do that, if you will do this." You will find that you will nicely bargain off all the items on the lists. Each of you will go away from the bargaining table secretly thinking you have gotten the best deal.

Thereafter, each will try to live up to his part of the bargains. If either breaks any bargain, he can be reminded of it by his mate (such reminders cannot be termed "nagging"). A secret signal can be devised to be used in public. Mutual breaking of bargains is a major setback and must be avoided.

Save the lists. You will find that you will frequently want to revise them. That is ok, as long as both partners agree. As time goes by, the lists will change and evolve. The lists will be revised so much that they will no longer resemble the original lists. It will become apparent that the items on the original lists were just the beginning and that the items on the later lists come closer and closer to the real issues. It is true of nearly every unhappy married couple that the issues which they think are the causes of their troubles never turn out to be the real ones at all. The bargaining lists will evolve naturally. You will ultimately arrive at the realization of the "core" problems. The core problems will be the last list revision. At that time you will feel the time has come to set the lists aside and forget about them. The relationship will be growing on its own.

To achieve the desired result through this process will take you months, perhaps as long as a year or so (though you will feel an immediate reduction in tensions). Expect that keeping the bargains will be very difficult at times. It will be especially difficult to adhere to the ground rules. Having to keep the bargains and ground rules will cause anxiety in you. Expect that; it is a sign that you are working with the problem. That anxiety will gradually drift away as the lists are revised and the process continues. Your pace will depend on your motivation.

Good luck, and remember: adhering to the ground rules is equally as important as the bargaining process. You will have nearly irresistible urges at times to break the ground rules. When the time comes to throw away the lists, you will find that the ground rules vanish. The ground rules will be part of you and you will no longer have to try to adhere to them. But do not expect perfection. You will have your lapses. No one is perfect.

Addendum

The method of using patient made problem lists to focus psychotherapy has been used successfully in a crisis oriented outpatient clinic by Rusk, Sternbach, and Goltz in the Department of Psychiatry at the University of California, La Jolla (unpublished material). The addition of bargaining and ground rules to such a process allows the system to be considerably more "self help."

R.F.D. 1, Union, Maine 04862

The Computer in Family Practice

CHARLES C. MORRISON, M.D., GEORGE W. BOSTWICK, M.D.
and SAMUEL L. BELKNAP, M.D.*

SYNOPSIS — ABSTRACT

Three years of experience with direct computer application in ambulatory medical care delivery is reviewed. The current setting is a three-man private family practice in rural Maine.

Emphasis is placed on multiple use of a time-shared computer access. A review is given of those applications which seem acceptable to physicians and patients along with comments on those that aren't.

Tables are provided on cost effectiveness which suggest that the time sharing computer may soon be an integral part of small private practices.

What is the computer's place in family medicine today? Is it on the horizon as a threat to the individualized personal medical care characteristic of the family practitioner or will it be an aid to him by which he may increase the quality and satisfaction of his practice? Regardless of the answers to these questions, many persons consider the $800,000 ro $14,000,000 investment that is commonly referred to as well beyond the overhead of the practicing physician's office. The purpose of this article is to put these and other questions on computer application into a realistic perspective. It is based on three years of experience with direct computer patient interactions both in the military and in private practice.

Background

Many reports of medical computer applications are based on adaptations of traditional commercial computer systems. The hardware and elaborate data processing equipment originally developed in response to business needs were used as a center for medical systems. Necessary new equipment was then crudely and expensively developed to be compatible with the traditional systems rather than the unique characteristics of the medical demand. The same was true of the "language" or "programming alphabets" in that medical practice was made to fit business language. The outcome, as might be expected, is the technological nightmare towards which medical literature is oriented.

The computer systems about which this article deals are those which lend themselves to medical application by meeting the following criteria:

1. relatively low cost,
2. extensive programming with medically compatible language,
3. terminal constellations which permit direct non-threatening patient interaction, and
4. no technological expertise required by office personnel.

Initial experience with such systems was obtained while developing the Programmed Patient Care Clinic at the Brunswick Naval Air Station in Brunswick, Maine. A project was begun in 1970 to provide solutions to the problems of medical records, mobile staff, mobile patient populations, and inefficient laboratory and x-ray utilization, by converting a large ambulatory population to the problem oriented record. It soon became apparent that the computer could do consistently what many human hands could also do — but in-consistently. Navy families at that installation have had no paper records maintained for about one year while their medical care has been logically recorded and stored by computer. Cost savings, physician satisfaction, and patient acceptance have been adequately demonstrated and will soon be submitted for publication.

In July 1971, our three-man private family practice began to integrate the computer in office practice. Initial funding for the computer terminal access was provided by the Department of Health and Welfare under the Computer Assistance for the Physician's Office Project (CAPO).

It was our contention from the outset that the computerized integrated medical record was a worthwhile goal. Equally obvious however was the fact that the computerized medical record would not by itself provide enough practice income to balance the increased overhead. The object of our efforts then became to convert as many of the administrative duties as possible to computer techniques as well as develop additional aids to patient care which would be economically supportive. Table 1 represents the status of these efforts. "Current Use" denotes daily office access since July 1971.

Patient Histories

An inevitable outcome of mixing physicians and computers is a medical history. Perhaps this is because history taking is an essential but very time consuming part of medical practice; perhaps it is simply an outlet by which a physician-author may express his latent creative talent. Our patient history which was developed after a process of analysis

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TABLE 1

Current Use	*Planned Additional Use*
1. patient history (physical exam format)	— individualized "take home" patient summary — insurance history
2. patient education	— expanded library — keyed to patient history
3. physician education (consultation)	— expanded library
4. paramedic guide for triage and examination	— refinement and remote implementation
5. office billing	— scheduling
6. diagnostic aids and reference scan	

and selection is no better or worse than most of the many other computer adapted medical histories. It is a branching general medical history which requires about 30-45 minutes of unassisted patient time. As have most computer oriented histories, it has been well received by patients and effectively utilized by those physicians who integrate it into their routines.

It has been quite obvious, however, that the patient has received little additional tangible benefit from the use of an automated history. As far as he is concerned, the doctor has always "asked some questions" when he got his "check-up." The patient is hardly motivated to pay a higher fee so that the doctor can have a machine do part of his work. The concept of an *individualized take-home patient summary* arose from the desire to provide the patient more of a return for his time, provide a medical service worthy of an additional fee, and to take greater advantage of the computer's potential.

The patient's summary is generated directly from his responses to a traditional medical question but contains material of individual interest to his health maintenance and for *his* use. The simplest example may be as follows:

Question to patient: "Do you smoke?" Yes/No

Patient response: "Yes"

Patient print out: "Did you realize that smoking increases your risk of sudden heart attack as well as lung cancer?"

The patient print out becomes more individualized as replies become related to previous questions, i.e.: "Did you realize that at *your* age and weight *your* chances of dying over the next ten years are increased 3 times by *your* present rate of smoking?"

A simple insurance history has been developed from a compilation of common questions on the many different insurance company forms. Acceptance by the individual companies has not yet been obtained.

Patient Education

This is a time consuming part of medical practice regardless of whether or not the physician is intuitively motivated. Many diseases (diabetes, arthritis, etc.) as well as family medical needs (contraception, well-baby care, etc.) are easily adapted to individualized patient use routines. The programs we are currently using in this area were not created by us but by other participants in the CAPO project.

A series of questions is presented which define a patient's specific problem; for example, the type of contraceptive desired. This is followed by interacting questions with the patient responding to what she already knows and the computer providing information of which she is not aware. At the conclusion, a short summary of the factual material of which she was not aware is presented for her to take home.

Physician Education

Several established routines solely for physician education have been available for our use. One program involves the National Board patient treatment method, whereby a patient is treated progressively according to the physician's judgement and the physician is criticized or commended for the merit of his choice. A second technique uses a short case history presentation followed by a series of treatment options. The patient progresses or fails appropriately, and the physician is given information as to why his treatment may be inappropriate.

The programs now available and recent attempts to increase the available library have had very low interest and utilization. There are many reasons for this but the most apparent explanation is simply that the *practicing* physician needs (and will use) education programs related to treatment of the patients he is seeing. He is less interested in the academic exercises of treating hypothetical patients.

The most useful approach to computerized physician education appears to be the "consultation" program whereby a physician enters active patient data and receives interpretation and references appropriate to what he had entered.

Paramedic Guides

The computer is uniquely reliable for reproducing accurately whatever has been stored in it. the old saw "garbage in — garbage out" captures this feature of the computer as being completely dependent on the human thought processes that initially told it what to do.

It is exactly this reliability in repetition that is probably the one commonly agreed-upon need in the application of paramedical personnel. Generally it is accepted that a non-physician can do many things which a physician would otherwise spend his time on, but problems arise on the selection and timing of such tasks. If a physician

could stand next to his assistant and point out, for example, that when a hypertensive patient who is taking thiazides complains of a sore toe a different procedure might be used than when the same complaint is heard from a diabetic. If this process could be done in several chronic diseases and if the single physician were replaced with a team of specialists in each specific chronic illness, it is quite probable that patients would receive better long term care. The same is true with regards to the identification and treatment of many acute illnesses.

This is exactly the philosophy of computer direction for paramedical personnel involved in patient care which can be most directly applied in private practice. The very well thought out protocols on hypertension, cardiovascular disease, and diabetes developed at Beth Israel Hospital and used in our practice are early prototypes. The format involves a short series of disease-related questions, a medication review and double check on patient's usage, a short directed physical examination, and a guide to indicated laboratory tests. The computer interaction in this case is via the physician's assistant with a one page print out provided for the physician review and concurrence or interruption. The program does provide very consecutive levels of patient referral: i.e., should the patient see a physician immediately, within a few days, or at sometime in the future.

Other protocols have been developed for equally secure consideration of acute illness. In general, these routines do not yet lend themselves to computer programming. The problem encountered here is that the physician, while evaluating a viral sore throat may be "ruling out" many potential illnesses of a more serious nature simply by having the patient before him. He will be doing the same thing with the multiple symptoms which the patient will complain of; he is either finding pain in the throat, headaches, and joint pain all consistent with his diagnosis of a viral sore throat or indicative of a need for further evaluation. It has been repeatedly shown that only the rare patient presents with a single complaint and the more questions he is asked the more extensive his symptomatology becomes. If a computerized acute illness screening program for paramedical use is to be of value to private family practice it must contain the following:

1. a highly selective series of screening questions providing a broad but rapidly discriminating patient history.
2. a library of selected, office based testing and physical exam procedures which the paramedic is directed to perform from the "endpoints" above.
3. a brief expenditure of patient and computer time. Several preliminary computer models meet the first two criteria but require the physician's assistant to repeatedly consult the patient and the computer, back and forth until a picture of the patient's illness is provided. This requires an expenditure of time by the patient and the computer which may be satisfactory to the academic institution but is not practical in private family practice.

The acute illness screen we have available for our use takes from three to fifteen minutes depending on the depth of patient's symptoms. The single encounter directs the physician's assistant to perform physical examination procedures or laboratory tests as appropriate to the patient's response. The print-out summarizes the patient's history and the tests which the assistant has been directed to perform. The results of the tests/procedures are manually entered after the patient and assistant have left the computer and before he sees the physician (if he does).

This routine is presently in rather crude form in our practice and reflects the practical demands of a family practice after a busy tourist season. Another summer will hopefully find us providing better medical care to the chronic problems of our regular patients while the use of the computer and medical assistants will help us to more safely evaluate the transient illnesses of seasonal visitors.

Office Billing

Administrative applications of the computer are likely to be the most attractive to practicing physicians. The grinding burden of paperwork which consumes perhaps a third of our time is an area that most physicians would gladly delegate to a machine. It is perhaps ironic that medical administrators have been using computer routines to simplify their duties for some time while those of us who have both administrative and patient care demands neglect such aids. Many obligations, including requests for records from other physicians, insurance and "third-party" forms, and consultation requests (and replies) will be automatic benefits if a computerized fully integrated medical record was now available. Since it is not, individual programs have to be developed where appropriate and where a direct financial influence on practice overhead can be demonstrated. Physicians should perhaps be a bit more demanding to those third-party carriers who like to deluge us with multiple forms and requests which could, with little effort, be made compatible via a single common form produced by a computer from daily office entries.

Automated billing is the simplest form of administrative computerization which is available

to the practicing physician. Currently, a single financial entry for each patient encounter is made by a medical secretary into the computer. The entry is made via the same terminal that is used at other times of the day by patients for purposes described previously. From the single entry is generated a monthly patient bill, a daily bookkeeping log, and an on-going record of the services our practice provides. The latter can be readily correlated to practice overhead and individual service expense.

Diagnostic Aids and Reference Scan

There are commercially available diagnostic programs by which a physician may list the symptoms, laboratory tests, or physical findings his patient may have and receive a list of diagnoses with varying levels of probability, including appropriate references for further study. We have had such a service available to us in our practice for several months and have used it very little. Although the theory justifying such programs seems valid, it is likely that they overlook a basic difference between academic medicine and private practice that has been earlier alluded to. The practicing physician would benefit from step-by-step consultation on the patient care problems presented to him, but is less interested in learning the syndrome's "proper name" for its own sake. Such diagnostic programs will likely be of more value and greater use to family physicians if they are geared to problem treatment rather thans syndrome labeling.

It should also be mentioned here that using a computer in practice requires a change in habit. The change required to use a computer for obtaining a diagnosis seems more difficult than the change required by the routines described earlier. This may explain our low utilization of such programs.

Cost

Computer services in our area utilizing all the programs previously discussed is currently available at about $1,200-$1,300/month.

This is broken down as shown in Table 2. "Computer Access" means the right to "plug-in" a computer located into a commercial supplier geographically remote from the office itself. The cost of this access generally includes a certain amount of individualized programming, plus a basic group of programs "owned" by the company.

The dedicated telephone line in our area represents approximately 160 miles of leased line from a computer in Cambridge, Massachusetts to our office in Damariscotta, Maine. Hardware* terminals come in many different shapes and forms. We have found that realistically the terminals used by the patient makes little difference; he is no more intimidated by a keyboard with 20 keys than he is with four colored buttons, provided he only has to use one of four choices. It is very hard to justify the added expense of light sensitive screens and pen light indicators popular in some areas. Technically there are differences in printer speed that may be summarized by saying that a more rapid speed is useful for hard (paper) copy than is received well by patients. Most printers can be easily adapted for either speed: therefore it is not a cost consideration.

TABLE 2

Cost	
Computer Access	$ 700
Dedicated Telephone Line	$ 300
Patient Screen Terminal	$ 130
Printer (Hard Copy) Terminal	$ 70
Total/Month	$1,200

TABLE 3

Reimbursement	
Patient History (60/month at $5)	$ 300
Patient Education (10/week at $5)	$ 200
Paramedic Routines (20/week at $5)	$ 400
Office Billing	$ 300
	$1,200

Who Pays the Bill?

Reference to Table 3 will provide support for the contention that computer services may soon be economically justifiable in private family practice. Office charges may vary around the country, but few can contest the conservative estimates provided.

Our office has completed 60-120 physical examinations per month. If a $5 charge is made for the preventive medicine patient history included as part of this, $300 would return to practice expenses.

The patient education routines could be reasonably considered the primary area of concern for many patient visits each week. Should the computer be used for ten visits each week at a charge of $5 per visit, approximately $200 would be accrued each month. Paramedic routines and their charges may be similarly explained with reference to the text above for the service provided.

A brief discussion of office billing is due with

*A lesson in computer terminology — Hardware refers to equipment. Software refers to the "thinking" or programming put into the machine.

respect to the local experience of our practice. A secretary in our area earns $100-$125 per week. While our practice was growing, a single bookkeeper was slowly but steadily falling behind the work generated by two physicians. With the addition of a third physician and computerized accounting, a medical secretary is now able to adequately handle the needs of the practice and will have the flexibility to absorb the work generated by a fourth physician after the first of the year. The $300 "generated" by the computer is a conservative estimate of the additional salary required had we not had computerized billing.

No reference is made to the physician education, diagnostic aid or other administrative programs which are beneficial to the practice, but cannot be easily credited against overhead expenses.

Patient, Physician and Computer

It should be re-emphasized that the programs described have been applied by regular medical and paramedical personnel with no particular computer training. The acceptance of the computer by the patient has been almost uniformly enthusiastic here or elsewhere. The strange, almost mystical fear of the computer by the physician is very difficult to understand when one becomes accustomed to thinking of it as little more than a very efficient secretary with a phenomenal memory.

The "Big Brother Syndrome" relating the computer to some kind of government surveillance is remotely conceivable only in the context of government involvement we currently see in other medical areas. Where physician initiative is lacking, a federal agency seems ready to fill the gap. The danger with government involvement in medical computer application does not lie with regulation as much as it does with government authorship of programs provided for practicing physicians which have not had significant family physician input.

Ongoing problems of computer application in private practice deal primarily with the logistics of scheduling and the continuous need for updating old habit patterns required when a new technology becomes available. When these problems become solved, the computer will become a useful tool in private family practice.

QUO VADIS, MEDICUS? — *Continued from Page 223*

phenomenon, and an unpleasant threat to many of us, who look back wistfully to a simpler age when the M.D. degree was carte blanche to a lifetime career.

The theory is certainly sound: weed out the deadwood and keep the rest on their toes. How to design fair tests for all sorts of different categories of physicians is, however, a most formidable set of problems. But it is a subject about which we all should be thinking, for the handwriting seems to be there on the wall: "Mene, Mene. . . ."

At the moment a (possible) shortage of physicians makes it impractical to withdraw a large number of them from practice for any reason. But it may not be too visionary to imagine a deliberate program to generate enough new graduates (perhaps with a "streamlined" curriculum) to flood the market and permit some sort of forced retirement of all doctors more than 20 years out of training — unless they could show very special qualifications or pass stiff examinations. The "retirees" might be allowed to engage in nursing, medical assistantships or administrative work — all under close supervision of licensed physicians.

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Precautions: Do periodic serum electrolyte and BUN determinations. Do periodic hematologic studies in cirrhotics with splenomegaly. Antihypertensive effects may be enhanced in postsympathectomy patients. The following may occur: hyperuricemia and gout, reversible nitrogen retention, decreasing alkali reserve with possible metabolic acidosis, hyperglycemia and glycosuria (diabetic insulin requirements may be altered), digitalis intoxication (in hypokalemia). Use cautiously in surgical patients. Concomitant use with antihypertensive agents may result in an additive hypotensive effect.

Adverse Reactions: Muscle cramps, weakness, dizziness, headache, dry mouth; anaphylaxis; rash, urticaria, photosensitivity, purpura, other dermatological conditions; nausea and vomiting (may indicate electrolyte imbalance), diarrhea, constipation, other gastrointestinal disturbances. Rarely, necrotizing vasculitis, paresthesias, icterus, pancreatitis, and xanthopsia have occurred with thiazides alone.

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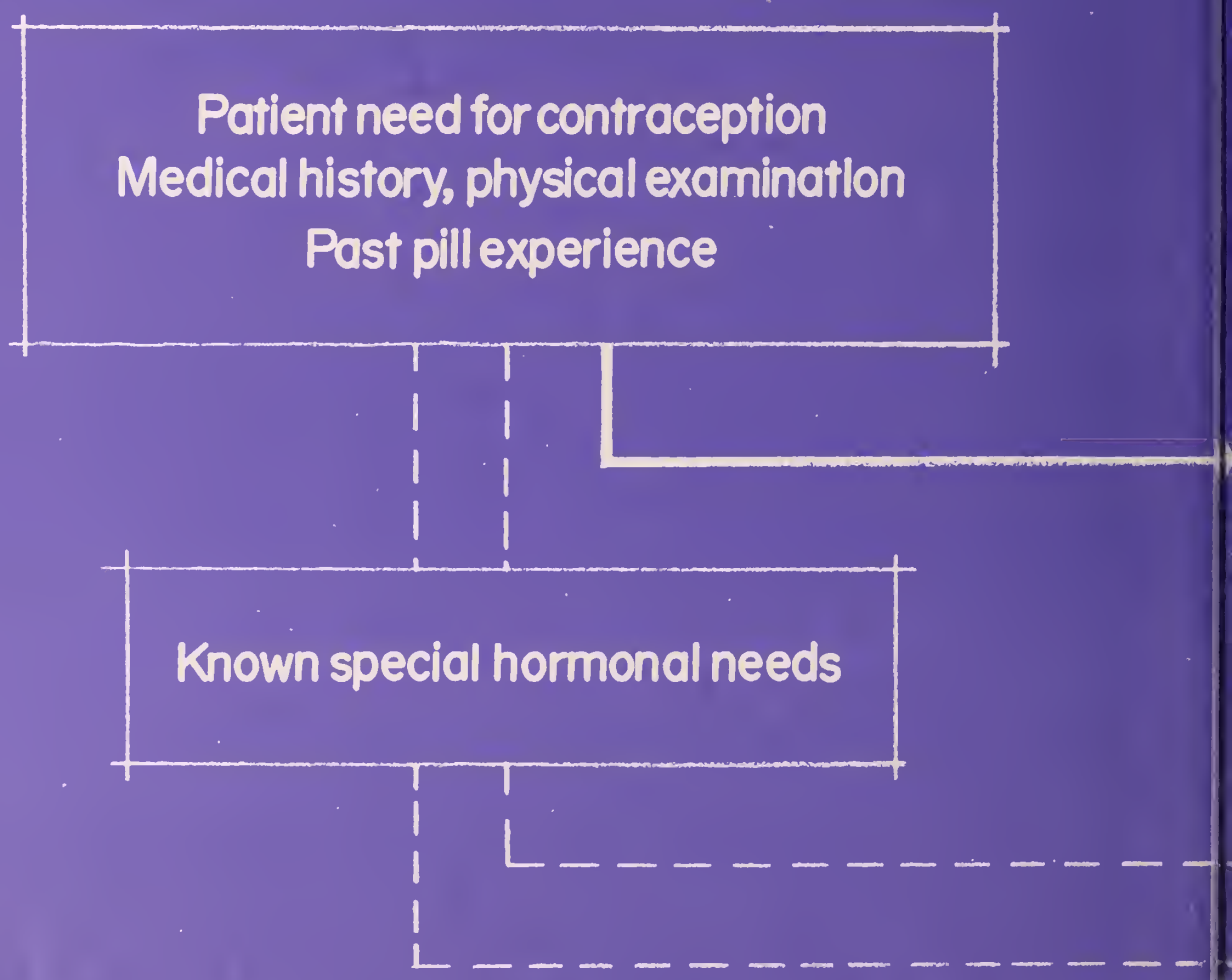

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Note: Oral contraceptives are complex medications. As with all medications they should be prescribed with discriminating care, and only after reference to full prescribing information. For brief summary of prescribing information, please see next page.

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Special note—Oral contraceptives have been marketed in the United States since 1960. Reported pregnancy rates vary from product to product. The effectiveness of the sequential products appears to be somewhat lower than that of the combination products. Both types provide almost completely effective contraception.

An increased risk of thromboembolic disease associated with the use of hormonal contraceptives has now been shown in studies conducted in both Great Britain and the United States. Other risks, such as those of elevated blood pressure, liver disease and reduced tolerance to carbohydrates, have not been quantitated with precision.

Long-term administration of both natural and synthetic estrogens in subprimate animal species in multiples of the human dose increases the frequency of some animal carcinomas. These data cannot be transposed directly to man. The possible carcinogenicity due to the estrogens can be neither affirmed nor refuted at this time. Close clinical surveillance of all women taking oral contraceptives must be continued.

Indication—Ovulen and Demulen are indicated for oral contraception.

Contraindications—Patients with thrombophlebitis, thromboembolic disorders, cerebral apoplexy or a past history of these conditions, markedly impaired liver function, known or suspected carcinoma of the breast, known or suspected estrogen-dependent neoplasia and undiagnosed abnormal genital bleeding.

Warnings—The physician should be alert to the earliest manifestations of thrombotic disorders (thrombophlebitis, cerebrovascular disorders, pulmonary embolism and retinal thrombosis). Should any of these occur or be suspected the drug should be discontinued immediately.

Retrospective studies of morbidity and mortality conducted in Great Britain and studies of morbidity in the United States have shown a statistically significant association between thrombophlebitis, pulmonary embolism, and cerebral thrombosis and embolism and the use of oral contraceptives. There have been three principal studies in Britain[1-3] leading to this conclusion, and one[4] in the United States. The estimate of the relative risk of thromboembolism in the study by Vessey and Doll[3] was about sevenfold, while Sartwell and associates[4] in the United States found a relative risk of 4.4, meaning that the users are several times as likely to undergo thromboembolic disease without evident cause as nonusers. The American study also indicated that the risk did not persist after discontinuation of administration and that it was not enhanced by long-continued administration. The American study was not designed to evaluate a difference between products. However, the study suggested that there might be an increased risk of thromboembolic disease in users of sequential products. This risk cannot be quantitated, and further studies to confirm this finding are desirable.

Discontinue medication pending examination if there is sudden partial or complete loss of vision, or if there is a sudden onset of proptosis, diplopia or migraine. If examination reveals papilledema or retinal vascular lesions medication should be withdrawn.

Since the safety of Ovulen and Demulen in pregnancy has not been demonstrated, it is recommended that for any patient who has missed two consecutive periods pregnancy should be ruled out before continuing the contraceptive regimen. If the patient has not adhered to the prescribed schedule the possibility of pregnancy should be considered at the time of the first missed period.

A small fraction of the hormonal agents in oral contraceptives has been identified in the milk of mothers receiving these drugs. The long-range effect to the nursing infant cannot be determined at this time.

Precautions—The pretreatment and periodic physical examinations should include special reference to the breasts and pelvic organs, including a Papanicolaou smear since estrogens have been known to produce tumors, some of them malignant, in five species of subprimate animals. Endocrine and possibly liver function tests may be affected by treatment with Ovulen or Demulen. Therefore, if such tests are abnormal in a patient taking Ovulen or Demulen, it is recommended that they be repeated after the drug has been withdrawn for two months. Under the influence of progestogen-estrogen preparations preexisting uterine fibromyomas may increase in size. Because these agents may cause some degree of fluid retention, conditions which might be influenced by this factor, such as epilepsy, migraine, asthma, cardiac or renal dysfunction, require careful observation. In breakthrough bleeding, and in all cases of irregular bleeding per vaginam, nonfunctional causes should be borne in mind. In undiagnosed bleeding per vaginam adequate diagnostic measures are indicated. Patients with a history of psychic depression should be carefully observed and the drug discontinued if the depression recurs to a serious degree. Any possible influence of prolonged Ovulen or Demulen therapy on pituitary, ovarian, adrenal, hepatic or uterine function awaits further study. A decrease in glucose tolerance has been observed in a significant percentage of patients on oral contraceptives. The mechanism of this decrease is obscure. For this reason, diabetic patients should be carefully observed while receiving Ovulen or Demulen therapy. The age of the patient constitutes no absolute limiting factor, although treatment with Ovulen or Demulen may mask the onset of the climacteric. The pathologist should be advised of Ovulen or Demulen therapy when relevant specimens are submitted. Susceptible women may experience an increase in blood pressure following administration of contraceptive steroids.

Adverse reactions observed in patients receiving oral contraceptives—A statistically significant association has been demonstrated between use of oral contraceptives and the following serious adverse reactions: thrombophlebitis, pulmonary embolism and cerebral thrombosis.

Although available evidence is suggestive of an association, such a relationship has been neither confirmed nor refuted for the following serious adverse reactions: neuro-ocular lesions, e.g., retinal thrombosis and optic neuritis.

The following adverse reactions are known to occur in patients receiving oral contraceptives: nausea, vomiting, gastrointestinal symptoms (such as abdominal cramps and bloating), breakthrough bleeding, spotting, change in menstrual flow, amenorrhea during and after treatment, edema, chloasma or melasma, breast changes (tenderness, enlargement and secretion), change in weight (increase or decrease), changes in cervical erosion and cervical secretions, suppression of lactation when given immediately post partum, cholestatic jaundice, migraine, rash (allergic), rise in blood pressure in susceptible individuals and mental depression.

Although the following adverse reactions have been reported in users of oral contraceptives, an association has been neither confirmed nor refuted: anovulation post treatment, premenstrual-like syndrome, changes in libido, changes in appetite, cystitis-like syndrome, headache, nervousness, dizziness, fatigue, backache, hirsutism, loss of scalp hair, erythema multiforme, erythema nodosum, hemorrhagic eruption and itching.

The following laboratory results may be altered by the use of oral contraceptives: hepatic function: increased sulfobromophthalein retention and other tests; coagulation tests: increase in prothrombin, Factors VII, VIII, IX and X; thyroid function: increase in PBI and butanol extractable protein bound iodine, and decrease in T^3 uptake values; metyrapone test and pregnanediol determination.

References: 1. Royal College of General Practitioners: Oral Contraception and Thrombo-Embolic Disease, J. Coll. Gen. Pract. *13*:267-279 (May) 1967. **2.** Inman, W. H. W., and Vessey, M. P.: Investigation of Deaths from Pulmonary, Coronary, and Cerebral Thrombosis and Embolism in Women of Child-Bearing Age, Brit. Med. J. 2:193-199 (April 27) 1968. **3.** Vessey, M. P., and Doll, R.: Investigation of Relation Between Use of Oral Contraceptives and Thromboembolic Disease. A Further Report, Brit. Med. J. 2:651-657 (June 14) 1969. **4.** Sartwell, P. E.; Masi, A. T.; Arthes, F. G.; Greene, G. R., and Smith, H. E.: Thromboembolism and Oral Contraceptives: An Epidemiologic Case-Control Study, Amer. J. Epidem. *90*:365-380 (Nov.) 1969.

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Indication—Enovid-E is indicated for oral contraception.

The *Special Note, Contraindications, Warnings, Precautions* and *Adverse Reactions* listed above for Ovulen and Demulen are applicable to Enovid-E and should be observed when prescribing Enovid-E.

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Reaction to Tetracycline?

ROWLAND B. FRENCH, M.D.

This is a report of a case of a patient with an unusual reaction to tetracycline therapy. It is repeatedly reported that drug reactions have become increasingly common, and have become a large source of difficulties in medical practice. In this area, penicillin reactions have been common, usually consisting of urticaria, wheezing and arthralgia with occasionally very severe allergic manifestations. One patient, in particular, developed severe urticaria and swollen joints requiring steroid therapy, whereupon the patient developed a psychosis secondary to the steroid, and was, between the two, disabled for months. Another patient was admitted in coma which developed one-half hour after ingestion of one penicillin tablet. He was pale, sweating, blood pressure 80/50, and his skin was a diffuse, fiery red color. This gradually improved over a one-day period, but he continued to have a severe headache, chills and slight itching.

CASE REPORT

The patient was a thirty-six-year-old male who was admitted to hospital for tooth extraction because of infected teeth. Past history revealed an ulcer operation ten years prior to entry. Laboratory: white blood count 5,500; hemoglobin 15 gm.; urinalysis normal. The patient had a tooth extraction under sodium pentothal anesthesia, receiving also quelicin and penthrane. Following the tooth extraction, the patient received Achromycin,® 250 mg., from the dentist, and after having three capsules at approximately four-hour intervals, he noted that his joints were slightly stiff and his muscles were not coordinating well.

The patient was discharged in the morning, but on the evening of the same day, he had noted, after continuing Achromycin at home, stiffness of his legs and difficulty getting up and was re-admitted to the hospital. Examination at time of readmission was essentially normal except for severe generalized muscle stiffness, with some tight feeling across the chest. He was able to move his arms, legs, chest and abdomen only with great difficulty. The most remarkable feature was severe stiffness of all muscles. Reflexes were normal. There was no Chvostek's sign, no trismus, no tremor. His neck was stiff but part of a generalized stiffness. He had no urticaria or joint swelling but some joint soreness and stiffness. His chest was clear. His heart was not enlarged and was regular with no murmur present. His abdomen showed generalized soreness of his abdominal muscles. Laboratory: white count 7,200 with normal differential but 1% eosinophils. Urinalysis was Sp. gr. 1013, occ wbc., alb. neg., sugar neg., small amount amor. phos. ph-6, hemoglobin 15.5 mg., sedimentation rate 15. The patient showed no sign of hyperventilation or nervous tension. Past history revealed the patient had infectious hepatitis in 1961; a duodenal ulcer operated in 1961; an undescended testicle operated at Bethesda Naval Hospital in 1953, at which time he states he had a penicillin reaction consisting of "being in coma at the Naval Hospital for approximately a two-month period." His medication was changed to Erythromycin for the tooth infection, and he improved on predisone 6 mg. qid. The patient was discharged after the stiffness had gradually disappeared. The predisone was gradually reduced. He had slight shaking at times, some stiffness of the left leg and felt tired, but at the end of a six-day period felt almost back to normal.

Toxic reactions to tetracycline have commonly produced gastrointestinal irritation. Less commonly have been reported hypersensitivity reactions including skin rashes, urticaria and more severe allergic symptoms. There also has been liver damage, phototoxicity, brown staining of teeth in children, increased intracranial pressure in infants and in adults and association of development of superinfections. In pregnancy, kidney and liver damage may occur as well as possible teratogenic effects. It may be dangerous to use tetracycline in the presence of decreased renal function. Outdated tetracycline has been reported associated with the Fanconi syndrome and simulated systemic lupus erythematosis.

The reported case had no urticaria, headache or visual disturbance. He had a slight wheeze, although his lungs were clear. No lumbar puncture was performed so the spinal fluid pressure is unknown. The history of previous penicillin reaction and slight wheeze would suggest that this was a hypersensitivity reaction. Previous case reports of sensitivity reaction to tetracycline have mentioned urticaria, breathing difficulties and collapse. This patient largely showed severe generalized muscle stiffness. (Recently the patient is said to have had a somewhat similar reaction following the use of pentothal at another hospital but details are unknown).

REFERENCES

1. Haas, R.: Anaphylactoid Reactions to Oral Tetracycline, J. Maine Med. Assoc., 48:85, March 1957.
2. Anaphylactic reactions to tetracycline: J.A.M.A. — Editorial — June 14, 1965, vol. 192, No. 11.
3. Fellner, M. J. and Baer, R. L.: Anaphylactic Reaction to Tetracycline in a Penicillin Allergic Patient, J.A.M.A., Vol. 192, No. 11, pp. 997-998.
4. Bedford, P. D.: Idiosyncrasy to Aureomycin, Brit. Med. J., 1:1428 (June 23) 1951.
5. Ilyas, M.: Hypersensitivity to Ampicillin and Tetracycline, Lancet, 2:417, 19 Aug. 67.
6. Garrod, L. P.: The Toxicity of Antibiotics, Med. J. Aust., 2:947-50, 12 Dec. 64.
7. Kagan, B. M. and Faller, L.: Untoward Reactions to Antibiotics, Med. Clinic N. A., 39:111, 1955.
8. Bibliography on Reactions to Tetracycline, Clin. Pharmacol. Ther., 7:126-32, Jan., Feb. 66.
9. Barnett, Crawford, F.: Anaphylactoid Reaction to Orally Administered Tetracycline, Southern Med. J., 60:963, Sept. 1963.
10. Koch-Weser J. and Gilmore, E.: Benign Intracranial Hypertension in an Adult after Tetracycline Therapy, J.A.M.A., 200:345-7, 24 April 1967.

16 Water St., Eastport, Maine 04631

From the Secretary's Notebook

Summary of 1973 Annual Meeting of the M.M.A. House of Delegates June 16 and 17 at Kennebunkport, Maine

The 120th annual session of the M.M.A. House of Delegates was held at the Shawmut Inn, Kennebunkport, Maine, with an attendance of seventy-six delegates and alternates, and forty-one guests. The first session was held on Saturday afternoon at 2:00 P.M., and the second session on Sunday afternoon at 2:00 P.M. George W. Wood, III, M.D., President of the M.M.A., called to order the meetings of the House, which were presided over by George W. Bostwick, M.D., Speaker of the House.

Reference Committees — Dr. Bostwick announced the procedure for the conducting of business this year — during the first session of the House, business will be introduced without debate and referred immediately to a Reference Committee. The Reference Committees will meet at staggered times on Sunday morning, where debate will take place. The Reference Committees will report to the second session of the House in the afternoon, and will make their recommendations. There will be open debate on any of these recommendations.

Election of Speaker and Vice Speaker of the House of Delegates for 1973-1974 — George W. Bostwick, M.D., was reelected Speaker of the House of Delegates and Richard W. Swengel, M.D., Vice Speaker of the House.

Budget for 1974 — The proposed budget for fiscal year 1974 was approved.

Committee on Nominations — The following slate was presented by this committee (Nelson P. Blackburn, M.D., Chairman):

President-Elect
John B. Madigan, M.D. and James C. Bates, M.D.

Executive Committee
1st District — Robert F. Ficker, M.D. and Willem F. Nieuwkerk, M.D.
5th District — H. Carl Amrein, M.D. and Carlton E. Swett, M.D.
6th District — William C. Bromley, M.D. and Robert F. MacBride, M.D.

Delegate to AMA
Robert E. McAfee, M.D. and
Daniel R. Shields, M.D.

Alternate to AMA
Brinton T. Darlington, M.D. and
Harry M. Helfrich, Jr., M.D.

Nominations from the floor included: Euclid M. Hanbury, Jr., M.D. and Richard T. Chamberlin, M.D. for President-elect. There were three written votes for President-elect because of ties. The following officers were elected:

President-Elect
John B. Madigan, M.D.

Executive Committee
1st District — Robert F. Ficker, M.D. (3 years)
5th District — H. Carl Amrein, M.D. (3 years)
6th District — William C. Bromley, M.D. (3 years)

AMA Delegate – 1/1/74 – 1/1/76
Robert E. McAfee, M.D.

AMA Alternate – 1/1/74 – 1/1/76
Brinton T. Darlington, M.D.

With Dr. Madigan's election to President-elect, a vacancy in the Ninth District occurred on the Executive Committee. The Committee on Nominations met briefly and brought in two nominations — Drs. Benoit Ouellette and Harry M. Helfrich, Jr. Dr. Eric Nicholas was nominated from the floor. Following a written ballot vote, Dr. Benoit Ouellette was elected to complete Dr. Madigan's term (until June of 1975).

The Standing Committees, as recommended by the Committee on Nominations, were approved as presented, with the addition of Dr. Floyd Goffin to a three-year term on the Peer Review Committee.

Dr. Blackburn, Chairman of the Committee on Nominations, stated that under the present bylaws, 2 candidates have to be presented, and this has created many problems because of the reluctance of

many to become involved in a "contest." Dr. Stitham reminded the delegates that the bylaws were just recently changed to require two, rather than one, to encourage more participation and to discourage the practice of becoming President by "going through the chairs." The Speaker of the House suggested that the Executive Committee review these comments at a future meeting.

Reports (not included in the House of Delegates folder) —

Executive Director — Dr. Daniel Hanley's report covered many subjects including new rules and regulations for health care, expenditures for health care, PSRO and Peer Review, Regional Blood Banks, Medical Education and Malpractice.

Committee on Legislation — Brinton T. Darlington, M.D., Chairman, reported this has been an extremely busy year, with much of their time and energy devoted to chiropractic and physicians' assistants bills.

Mr. Charles L. Cragin, III, M.M.A. lobbyist for this past legislative year, gave an excellent presentation on what lobbying is all about. He suggested that physicians should take a more active interest in politics, take a position on all health care legislation in general, and turn out at a hearing when asked. A summary report on how our Association fared during this session will be submitted in the near future, Mr. Cragin added.

Diabetes Committee — Melvin Bacon, M.D., Chairman (copy of report sent to members of the House of Delegates and County Society Presidents).

Peer Review — Richard T. Chamberlin, M.D., Chairman, reminded the delegates of their past actions in regard to Peer Review, and posed several questions to the delegates that still remain in question. In concluding, he asked, "just where does Maine stand? Your Peer Review Committee would like to know." The Reference Committee suggested that this report be referred to the Executive Committee for study, and it was so *voted.*

RESOLUTIONS

Chiropractic in State Services — This resolution, submitted by the Penobscot County Medical Society, resolving "that the Maine Medical Association recommends that the State of Maine Department of Health and Welfare authorize treatment by chiropractors only when such services are ordered by a physician who is responsible for both the physical care of the patient receiving such services and the determination of the efficacy and need for continuation of such services," and further resolving "that the Association recommends that payment by the State of Maine Department of Health and Welfare or by its Division of Accounts and Audits for chiropractic services be made only when such services have been ordered by a physician, and after the Department has determined that a patient receiving such services is under the care of the physician who ordered such services," was *postponed indefinitely.*

Medicredit — Submitted by the Executive Committee, this resolution was amended and *approved* as follows:

WHEREAS, we believe that health insurance programs, private or public, should enable every person to obtain insurance protection which would make available to him personal health services from professionally qualified providers of his choice; and

WHEREAS, the standards of coverage for all insurance and prepayment programs approved for enrollment should be uniform, the coverages comprehensive, and the benefits and premium charges realistic; and

WHEREAS, the principle of "Individual Responsibility" may be implemented through the mechanisms of co-insurance and deductible features; and

WHEREAS, recognition of ability of certain persons allows the more economical use of tax funds to provide coverage for those unable, or less able to pay;

NOW, THEREFORE, BE IT RESOLVED: That the House of Delegates of the Maine Medical Association endorses in principle, the basic concepts stated above and notes that the *AMA Medicredit Bill* most closely embodies these ideals. The House of Delegates of the Maine Medical Association urges the continued search for innovative fiscally responsible health care financing plans.

Continuing Education — This resolution, submitted by the Committee on Continuing Education, was unanimously approved:

WHEREAS, the House of Delegates of the Maine Medical Association passed a resolution in June of 1972 which charged the Continuing Education Committee and the Executive Committee of the Maine Medical Association to define "the exact educational requirement which shall be necessary for continued membership in this society in the future so that appropriate bylaw changes may be properly developed and circulated . . . no later than June of 1973," and

WHEREAS, the Continuing Education Committee has studied the various methods used throughout the country to accomplish

similar goals, and

WHEREAS, the initial experience with an educational requirement in other state medical associations has been favorable, and yet

WHEREAS, it has been difficult to show proof that specific requirements for a given number of hours of continuing education guarantees that the participant has, in truth, actually learned anything, or improved the care given to patients, and

WHEREAS, we in Maine have little or no concept of what our membership is currently doing on a voluntary basis for their own continuing medical education needs, and

WHEREAS, such information might help the Maine Medical Association in formulation of future requirements, now therefore

BE IT RESOLVED, that the Maine Medical Association Bylaws be amended as follows:

Chapter 1. Membership. Add a new section. Section 11. Each member of the Maine Medical Association shall be required to report his activities in continuing medical education every year starting with 1 January 1974 according to a format provided by the Committee on Continuing Medical Education.

Chapter V. Executive Committee. Duties. Section 3. After the words "individual Director" in the next to the last sentence, add the following sentence, "The Committee may also suspend or place a member in a probationary status for failure to comply with the continuing education program of the Association or failure to submit reports satisfactory to the committee showing compliance with said program."

Chapter VII. Councils and Committees. Section 4. Council on Health Manpower and Education. Section D. Committee on Continuing Education. After the four methods defined in the present bylaws, add the following paragraph. "The Committee shall collect and study the annual reports of the efforts in continuing medical education of physician members of the association and make recommendations to the Executive Committee about the membership status of the individual members with regard to the continuing educational reporting requirement Chapter 1, Section 11, above."

Blood Banking — Submitted by the Executive Committee, this resolution was *approved:*

WHEREAS, the concept of Regional Blood Banking offers the people of the State of Maine improved availability of blood products.

THEREFORE, BE IT RESOLVED, that the concept of Regional Blood Banking be endorsed by the Maine Medical Association.

Endorsement of Statewide Data Collecting Agency — This resolution from the Cumberland County Medical Society was *amended* and *approved*:

WHEREAS, the State of Maine as represented by the State Legislature and the Department of Health and Welfare, has established the need and desirability of a central State Tumor Registry, and

WHEREAS, a Tumor Registry should record incidence of cancers, their staging, their modes of treatment, should maintain the follow-up and ultimately record and publish survival data, and

WHEREAS, for many years the American College of Surgeons has approved cancer programs in many hospitals in the State of Maine, whose program is the Tumor Registry, and

WHEREAS, ongoing professional education in cancer is an integral part of a successful cancer program and a useful cancer registry reflects the educational program, and

WHEREAS, recent congressional legislation requires that data be collected relating to norms and efficacy of medical management, now therefore

BE IT RESOLVED, that as a beginning to the solution of these several problems, the Maine Medical Association endorse the concept of a central Statewide data collecting service and to encourage the rapid implementation of this service as soon as possible.

Committee on Maternal and Child Welfare — #1 — This resolution, as submitted, was *approved:*

WHEREAS, it is recognized that the nation's most valuable resource is its children, and

WHEREAS, it is recommended that every infant receive quality medical supervision in the earliest period of life, and

WHEREAS, the private insurance industry, with the exception of Blue Shield, offers exclusion from coverage for the first fourteen days of life in many policies, and

WHEREAS, certain health problems in children are readily identified and best treated in the first days of life, and consequently, this exclusion is not for the benefit of child health nor the family's well-being,

THEREFORE, be it resolved that the Maine Medical Association vigorously supports the eradication of the fourteen day exclusion clause from all health insurance policies sold in the State, urges the Insurance Commissioner of the State of Maine to make it mandatory to include care of newborns for the first fourteen days of life in all health insurance contracts issued in this State, and, furthermore, if legislation is necessary, that the legislature of the State of Maine enact the proper legislation to give this coverage to newborns during the first fourteen days of life in health insurance policies sold in this State.

Committee on Maternal and Child Welfare — #2 — After considerable discussion, this long resolution re treatment of minors, was amended and referred to the Executive Committee for study and then for presentation at the next meeting of the House of Delegates.

Professional Standards Review Organizations (PSRO's) — This resolution, submitted by the Lincoln-Sagadahoc County Medical Society, resolving that the M.M.A. affirm a policy to forbid any contractual arrangement. . . , was *defeated,* and an alternate resolution submitted by the reference committee. After considerable discussion, this resolution was *approved* as follows:

WHEREAS, Public Law 92-603 has been enacted, and

WHEREAS, specific details of the guidelines are not yet available, and

WHEREAS, we could comply with legislation which allows:

a) Freedom of choice between physician and patient
b) Freedom of choice of mode of therapy
c) Ability to maintain the confidentiality of the record of any patient's illness
d) Liberal consideration of the welfare of the patient and reasonable clinical research, and

WHEREAS, the P.L. 92-603 will be implemented by other agencies than doctors after January 1st 1976 unless physicians take an active part, and

WHEREAS, physicians in the State of Maine reaffirm the concept of peer review,

THEREFORE, BE IT RESOLVED, with above qualifications, that members of the Maine Medical Association be in qualified compliance with P.L. 92-603, and that the President be directed to instruct the Executive Committee to study in depth P.L. 92-603, and this committee shall be instructed to present to the next meeting of the House of Delegates their recommendations for action.

Maine as the PSRO Area — Submitted by Dr. Richard T. Chamberlin, this resolution re designating Maine as the PSRO area, was *defeated.* An additional resolution was presented by Dr. Wood who said he hoped it reflected the viewpoint of the members of the Association. He attempted to say what the Chamberlin resolution said, but also said there appears to be inadequacies in the law and we should help to re-write it. The resolution as *approved* reads:

WHEREAS, PSRO, as defined in Title II of Public Law 92-603 has been passed by the Congress in October 1972 and,

WHEREAS, the goals of PSRO are at best difficult to achieve and,

WHEREAS, the practicing physicians are best equipped to define the guidelines and regulations in concert with the appropriate agencies of HEW and,

WHEREAS, as noted in Section 1152, licensed physicians in an area may be organized to perform professional reviews and evaluations of patient care services for that area, and

WHEREAS, the practicing physicians of the State of Maine should speak as one voice in advising the Secretary of HEW of its positions,

THEREFORE, BE IT RESOLVED:

1. The Maine Medical Association advise the PSRO section of HEW of the deficiencies in Title II of Public Law 92-603.
2. That the membership of the M.M.A. work with officials of PSRO (HEW), American Medical Association, and other state medical associations to correct these deficiencies.
3. That the Secretary of HEW be informed that in order to be most effective, the State of Maine should be designated as a single PSRO area.

Pediatric Nurse Associate Statement of Policy — A copy of this statement was given to each delegate and it was *approved.* The reference committee suggested that more concrete policies of evaluation be devised by the Liaison Committee, and it was further suggested that a report on each assistant be given every six to twelve months for the first five years of the program. These suggestions were approved by the House.

President of the Woman's Auxiliary to the M.M.A. — A report from Mrs. Paul A. Brinkman was read by Mrs. Robert LaFond.

Maine Association of Medical Assistants — Ms. Betsy Jo Whitcomb, President, informed the delegates that their members are more than just secretaries. In Portland, they run a registry which screens medical assistants, and maintains a list of available assistants at all times. They are also involved in a certification program whereby one must pass an exam which covers anatomy, secretarial and administrative practices, lab procedures, etc. Ms. Whitcomb added that she hopes the physicians will encourage their secretaries to join their organization, and to take the certification exam.

Maine Blue Cross and Blue Shield Award of Appreciation — This award was presented to Daniel F. Hanley, M.D. of Brunswick by Mr. Richard F. Nellson, President of Maine BCBS.

A. H. Robins Company Community Service Award — The Community Service Award for 1973 was presented to E. Stanley Young, M.D. of Poland Spring.

New England Physicians Retirement Program — Mr. John J. Casey and Mr. David Gerstenblatt explained the services provided by this program and briefly explained the financial advantages available to a physician who incorporates. A slide presentation comparing the Keogh Plan versus corporate was given.

University of Vermont Visiting Practitioner Program — Dr. J. Donald Schultz, Coordinator of this program, explained how it is individually structured for practicing physicians according to the expressed needs of the physician. They will arrange your schedule to teach you what you want to learn, during the time period you have selected. He described the program as a unique, individualized postgraduate experience which functions as both an educational and communicative program.

Family Practice Residency Program in Portland — Dr. Darrell P. Thorpe explained that the Maine Medical Center has had a very successful GP residency program for over 10 years, but it has become obvious that the name of the game is now "family practice" and the MMC has been looking for a way to make the conversion. Area Health Education Centers (AHEC) came into being, and they (MMC) build their contract proposal around a Family Practice Residency Program. The major difference in training for an FP as opposed to GP is the emphasis on outpatient activity, ambulatory care activities, and this is structured around a model family practice unit. A low-income section of Portland, Munjoy Hill, will be the location for the model family practice unit.

Fee Schedules — Dr. Paul A. Fichtner recalled that a year ago, the House of Delegates adopted a resolution which approved a uniform, State-wide fee schedule. Following through on this, Dr. Fichtner visited Union Mutual (Medicare) where physician profiles are determined. After some time, Union Mutual agreed to do a mini-profile to determine the feasibility of converting to a uniform fee schedule for the State (it was learned that Maine is divided into 3 areas now). The decision from Union Mutual was that they could not go to a single fee schedule as it would result in a rollback of allowable charges for some procedures in various areas.

Tumor Registry — Dr. Ronald Carroll of Portland, Director of Oncology, reported that tumor registries are not new, but functional ones are uncommon, if not non-existent. Two years ago, the State of Maine made cancer by law a reportable disease. The State has sought to establish a State Tumor Registry and that was the fundamental reason for establishing cancer as a reportable disease. The registry should record the incidence of cancer, staging of cancer, modes of therapy of cancer, and ultimately publish survival statistics and document the magnitude of the cancer program in the communities for the local physicians, Dr. Carroll added. He presented slides of a model tumor registry.

Central Maine Family Practice Residency Program — Dr. Robert Ohler reported the purposes of this program to be: 1) there is a lot of capability in Central Maine for house staff training, and 2) in Central Maine, there is a real need for primary physicians. The first step is to develop house staff training capabilities. They will start with 3 residents on July 1st, and they will eventually be able to take 6 residents a year, in each of the 3 classes, for a total of 18 residents. This program is being financed in large part by the Veterans Administration. A combined effort of Central Maine General, Augusta General, St. Mary's, Thayer and Togus, this program is unique because nowhere is there such an effort that uses five hospitals, Dr. Ohler added.

Report of Reference Committees — All printed and oral reports referred to these three committees were approved. It was suggested that Executive Committee reports be received from *all* the districts, and that they be more complete. The Legislative Committee and our lobbyist, Mr. Cragin, were commended for their efforts and the hope was expressed that Mr. Cragin be continued as lobbyist, but no vote was taken. It was recommended to the Committee on Recruitment, Aid & Placement that more effort be made to encourage preceptorships.

Out-of-State Delegates and Guests — The following guests were introduced: Dr. John E. Farrell of Rhode Island; Dr. Thomas McCarthy of New York; Dr. Harry Rowe of Vermont; Dr. Thomas G. Cogswell of New Hampshire; Dr. Bernard O. Nemoitin of Connecticut; and Dr. Norman Gardner, VP of AMA. Dr. Cogswell suggested a combined New Hampshire-Vermont-Maine meeting in Montreal in 1975, and this was referred to the Executive Committee for consideration.

Special Memberships — The recommendations for special memberships were approved, with the transfer of Dr. Philip Archambault from Affiliate back to Active.

Miscellaneous —
The Reference Committees were commended for

Continued on Page 242

Maine Medical Association

STANDING COMMITTEES — 1973-1974

Standing Committees for 1973-1974 as proposed by the Committee on Nominations and approved at the Second Meeting of the House of Delegates of the Maine Medical Association at Kennebunkport, Maine, June 17, 1973.

Council on Health Manpower and Education

Committee on Allied Health Professions

William L. MacVane, Jr., M.D., 211 State St., Portland 04101 (1 yr.) — Chairman
George W. Hallett, M.D., 22 Bramhall St., Portland 04102 (1 yr.)
Robert G. MacBride, M.D., 25 Washington St., Lubec 04652 (2 yrs.)
George W. Bostwick, M.D., P.O. Box 388, Newcastle 04553 (3 yrs.)
Christopher S. Smith, M.D., P.O. Box 232, Farmington 04938 (3 yrs.)

Committee on Continuing Education

Richard T. Chamberlin, M.D., Thayer Hospital, Waterville 04901 (1 yr.) — Chairman
Robert H. Pawle, M.D., 251 U. S. Rt. 1, Falmouth 04105 (1 yr.)
Charles W. Capron, M.D., 22 Bramhall St., Portland 04102 (2 yrs.)
Floyd B. Goffin, M.D., Pennellville Rd., Brunswick 04011 (3 yrs.)
Henry J. Wheelwright, M.D., Augusta General Hospital, Augusta 04330 (3 yrs.)

Committee on Recruitment, Aid and Placement

Paul A. Brinkman, M.D., Farmington 04938 (1 yr.) — Chairman
Robert E. McAfee, M.D., 7 Bramhall St., Portland 04102 (2 yrs.)
Herbert C. Gilman, M.D., 200 Spruce St., Millinocket 04462 (2 yrs.)
Charles H. Lightbody, M.D., No. Main St., Guilford 04443 (3 yrs.)
C. Philip Lape, M.D., R.F.D. #1, Orr's Island 04066 (3 yrs.)

Committee on Scientific Programs

Alan W. Boone, M.D., 111 State St., Bangor 04401 (1 yr.) — Chairman
Bradley E. Brownlow, M.D., Blue Hill Mem. Hospital, Blue Hill 04614 (2 yrs.)
Robert H. Pawle, M.D., 251 U. S. Rt. #1, Falmouth 04105 (3 yrs.)

Council on Medical Services

Committee on Care of the Disadvantaged

Stephen A. Sokol, M.D., Central Maine Gen. Hospital, Lewiston 04240 (3 yrs.) — Chairman
David C. Dixon, M.D., Box 792, Farmington 04938 (3 yrs.)
Melvin Bacon, M.D., 27 June St., Sanford 04073 (1 yr.)
James C. Bates, M.D., Eastport 04631 (1 yr.)
Gretl J. Hoch, M.D., Phillips 04966 (2 yrs.)
John J. Pearson, M.D., 100 So. Main St., Old Town 04468 (2 yrs.)

Committee on Emergency Medical Service

Darrell P. Thorpe, M.D., Maine Medical Center, Portland 04102 (1 yr.) — Chairman
Edward K. Morse, M.D., 22 White St., Rockland 04841 (1 yr.)
John W. Towne, M.D., 325C Kennedy Mem. Dr., Waterville 04901 (2 yrs.)
Winford C. Adams, M.D., 14 Starlight Dr., Brewer 04412 (3 yrs.)
Owen O. Dow, M.D., Box 388, Longwood Dr., Kennebunk 04043 (3 yrs.)

Committee on Government Health Activities

John H. Steeves, M.D., Route #3, Skowhegan 04976 (3 yrs.) — Chairman
Linus J. Stitham, M.D., 50 Main St., Dover-Foxcroft 04426 (3 yrs.)
Thomas F. Shields, M.D., 300 Pine St., Lewiston 04240 (1 yr.)
John R. Davy, M.D., 73 Deering St., Portland 04101 (1 yr.)
Walter L. H. Hall, M.D., 130 Middle St., Old Town 04468 (2 yrs.)

Committee on Health Care Financing

Euclid M. Hanbury, Jr., M.D., Medical Bldg., Belfast 04915 (3 yrs.) —(Waldo) — Chairman
Charles W. Steele, M.D., 472 Main St., Lewiston 04240 (2 yrs.) — (Androscoggin)
Gordon N. Johnson, M.D., P.O. Box 86, Houlton 04730 (2 yrs.) — (Aroostook)
Ferris S. Ray, M.D., 7 Bramhall St., Portland 04102 (3 yrs.) — (Cumberland)
Gaetano T. Fiorica, M.D., 12 Church St., Chisholm 04222 (1 yr.) — (Franklin)
John D. McIntyre, M.D., 50 Union St., Ellsworth 04605 (2 yrs.) — (Hancock)
Paul H. Pfeiffer, M.D., Cherry Hill Ter., Waterville 04901 (2 yrs.) — (Kennebec)
Oram R. Lawry, Jr., M.D., 96 Limerock St., Rockland 04841 (2 yrs.) — (Knox)
Francis A. Winchenbach, M.D., 910 Washington St., Bath 04530 (3 yrs.) — (Lincoln-Sagadahoc)
Adwaita K. Ganguli, M.D., 191 Lincoln Ave., Rumford 04276 (1 yr.) — (Oxford)

Thornton W. Merriam, Jr., M.D., 431 State St., Bangor 04401 (1 yr.) — (Penobscot)
Charles H. Lightbody, M.D., No. Main St., Guilford 04443 (3 yrs.) — (Piscataquis)
Harland G. Turner, M.D., Box 38, Norridgewock 04957 (1 yr.) — (Somerset)
John Kazutow, M.D., Box 113, Columbia Falls 04623 (1 yr.) — (Washington)
Alvin A. Hoffman, M.D., P.O. Box 38, York 03909 (3 yrs.) — (York)

Members of the Advisory Committee to the Committee on Health Care Financing

Maine Society of Anesthesiology — George W. Bostwick, M.D., P.O. Box 388, Newcastle 04553
Maine Chapter, American Academy of Family Physicians — John D. Denison, M.D., 89 Hospital St., Augusta 04330
Maine Society of Obstetrics and Gynecology — E. Allan McLean, M.D., 29 Deering St., Portland 04101
Maine Chapter, American Academy of Pediatrics — Everett A. Orbeton, M.D., 131 Chadwick St., Portland 04102
Maine Society of Internal Medicine (Includes Medical Specialty Group) — Albert Aranson, M.D., Maine Medical Center, Portland 04102
Section on Ophthalmology of the M.M.A. — Jay K. Osler, M.D., 74 Birch St., Bangor 04401
Maine Radiological Society — John F. Gibbons, M.D., 22 Bramhall St., Portland 04102
Maine Chapter, American College of Surgeons — John F. Reynolds, M.D., 325 Kennedy Mem. Dr., Waterville 04901
Ear, Nose and Throat Group — Loring W. Pratt, M.D., 325 Kennedy Mem. Dr., Waterville 04901
Maine Society of Pathologists — Franklin F. Ferguson, M.D., 22 Bramhall St., Portland 04102
Neurology and Neurosurgery Group — Daniel A. Rock, M.D., 477 Main St., Lewiston 04240
Maine Trauma Committee — H. Carl Amrein, M.D., 29 Weston Ave., Madison 04950
Maine Psychiatric Association — Aldo F. Llorente, M.D., 56 Baribeau Dr., Brunswick 04011
Maine Academy of Orthopedic Surgeons — Allan J. Stinchfield, M.D., P. O. Box 343, Augusta 04330

Committee on Hospital Association Liaison

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DEAN H. FISHER, M.D.
COMMISSIONER

State Of Maine

Department of Health and Welfare

Immunizations and Other Medical Considerations for International Travel

PETER J. LEADLEY, M.D.*

A great deal of confusion exists about the recommendations and/or requirements that are imposed upon persons travelling to other countries by certain health authorities with regard to immunizations and other health matters.

First, it is extremely important to differentiate between "recommendations" and "requirements." Specifically, "requirements" are those regulations which are established by the official health agencies of the United States and other countries and which relate to those immunizations that people must have when travelling to, or from, the country in question. These have the force of law. By international agreement, countries may issue "requirements" only about immunizations for smallpox, yellow fever, cholera and plague. As long as these international agreements remain intact, countries will not, and can not, require immunizations for other illnesses. On the contrary, many health agencies (local or city health departments, state health departments, medical associations, the Federal Government, travel agencies and others) issue "recommendations" to persons who are travelling. These recommendations are *not* consistent from one place to the other, nor from one time to the other, nor from one agency to the other. It is generally because of differences in these "recommendations" that confusion exists. Also, much confusion exists because people do not understand the difference between requirements and recommendations. Requirements must be followed — recommendations do not have to be followed.

Until recently, smallpox vaccination was required for all forms of international travel. In the course of the past decade, however, these requirements have been relaxed and now travellers, in most parts of the world, (with the exception of certain parts of Africa, So. America and the Indian subcontinent) are not required to have smallpox vaccinations for re-entry into the United States. Most other countries have similar requirements regarding smallpox at this time. Yellow Fever vaccinations are still required for travel to certain countries in Africa and So. America. Cholera vaccinations are still required for travel to certain countries in the Near East, in the Indian sub-continent, in Africa and occasionally for some countries in Southeastern Europe.

As was mentioned above, these requirements are established by the United States Public Health Service — Quarantine Division — and the best source of current up-to-date information about these requirements is the United States Center for Disease Control located in Atlanta, Georgia. This Center regularly distributes information about these requirements to the State Bureau of Health and anyone wishing specific information about these requirements can contact the State Bureau of Health for further information. At the present time and for the forseeable future, anyone planning to travel to Canada, Mexico, Central America, Europe, Eastern Europe and the Soviet Union, Hawaii, Japan, Australia, New Zealand and most of North Africa is not *required* to have any immunizations.

It is important to remember that these "requirements" can be changed at any time and can be changed very quickly, not only by this country, but also by other countries. Therefore, even though smallpox vaccination is not "required" for international travel, the Maine State Bureau of Health still "recommends" that travellers have this immunization within three years of the time of their travel. This recommendation is made to assure that travellers will be spared any inconvenience that they might otherwise incur because of sudden and unexpected changes in these requirements. We do not recommend any similar

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precautions for Yellow Fever or Cholera vaccination.

There are other "recommendations" which people contemplating international travel should keep in mind. Poliomyelitis still does occur in other countries and the Maine Bureau of Health recommends that anyone planning international travel, and is thirty years of age or younger, check with their physician to make sure their polio immunizations are complete. If they are not complete, we strongly suggest they be completed to preclude the possibility of polio being contracted during travel to other countries.

Since the facilities for medical care are sometimes difficult to locate or difficult to use in other countries, we also recommend that anyone contemplating international travel have their tetanus immunization status reviewed and updated. Anyone who has had a tetanus immunization within five years can be considered well enough protected for this purpose. Certainly, anyone who has never been immunized against tetanus should have a primary tetanus series, not just because of international travel, but also for general health protection.

Children who are accompanying their parents on international travel should have all their current immunizations up-to-date. Parents should check with their family doctor to make sure their children have current immunizations.

Persons travelling to areas where malaria occurs such as Africa, the Indian sub-continent, Southeast Asia, South America and Central America are advised to take certain medications on a weekly basis while they are in these countries to prevent the development of malaria. The exact type and frequency of medication should be determined by your family doctor and he should be consulted for information about this protective measure.

Persons who are going to be living under primitive conditions while abroad such as Peace Corps volunteers who will be living in native villages, people doing archeological or geological surveys in primitive areas, and others who will be engaging in similar kinds of activities while abroad should be considered for gamma-globulin prophylaxis against hepatitis. The gamma-globulin injections for this purpose are good for approximately six months and do provide some protection against infectious hepatitis. People who are travelling for pleasure and staying in the usual tourist accommodations generally do not need this form of protection because water supply and sewerage standards in other countries have reached the point where hepatitis is not a major problem in these types of facilities.

It should be emphasized that there can be no substitute for using good sense when travelling to avoid illness. When water supplies are questionable, boiling water is advised for at least twenty minutes to purify it before drinking. When boiling is impossible, bottled soft drinks, alcohol containing beverages such as beer and wine, and canned or bottled sterilized water is advised. Travellers are advised to avoid fresh vegetables and fruits which have not been carefully washed in clean water and also to avoid eating meats which have not been thoroughly cooked. These are common sense health rules which also apply in the United States as well as in foreign countries.

So called "travellers diarrhea" is a common problem for which there is no known cause at present. The best available medical research indicates that there are probably a number of causes for this condition which generally manifests itself as abdominal cramps, diarrhea, loss of appetite and feelings of weakness and lethargy. Usually fever is not present. A small number of people also suffer from nausea and vomiting with this problem. Certainly, travellers who know they are prone to develop so-called "travellers diarrhea" are well advised to check with their family doctor before leaving and obtain a small supply of medication to take should this problem present itself. Usually, the illness lasts no more than several days and is not particularly severe although it is quite troublesome. Any illness which lasts longer and is accompanied by high fever, or is otherwise more serious, should be regarded with suspicion and medical assistance should be obtained. Travellers are cautioned to avoid medications purchased in other countries for this condition as many of the medications commonly available have been banned in the United States because they are unsafe. Their effectiveness in treating this condition is also very questionable. Medications provided in this country by family doctors are the best treatment.

People who have regular need for certain medications are also advised to obtain a supply of their medication in this country and carry it with them. Many medications are available in other countries but their quality and dosage strengths vary quite significantly from U.S. drugs. If it does become necessary to purchase medication in another country, a physician's advice should be sought first. Also, because of the current interest and concern about drug abuse, travellers who need to carry certain medication such as sleeping pills, anti-convulsive medications for epilepsy, weight reduction medications, tranquilizers, or drugs for "nerves" and ulcer medications are advised to obtain a signed note from their physician indicating the type of medication they are carrying and the dose they take. It should also

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MANY PSRO PROVISIONS INCUMBENT UPON REGULATIONS

Bernard R. Tresnowski of the Blue Cross Association spoke at a joint session of the Blue Cross Association and the National Association of Blue Shield Plans about specific points in the Professional Standards Review Organization program which depend upon government regulation for implementation.

Mr. Tresnowski stated that PSRO legislation has been summarized, analyzed, highlighted, and interpreted broadly. Everyone has received many documents describing the legislation and its objectives, he observed, and noted that he was not going to do that: the law is there to be read. In net, we now all understand the role and function of the National Professional Standards Review Council (NPSRC), the State Professional Standards Review Council (SPSRC) and the Professional Standards Review Organization (PSRO). We also now understand the appellate opportunities for the beneficiary of services under one of the titles of the Social Security Act, and for the practitioner and provider rendering the services under these titles.

We are similarly aware of the statutory authorizations granted to the PSRO in the conduct of its functions as well as the implementation provisions in the Statute. We do not know what the Secretary's regulations will be in the many aspects of the PSRO provisions which require his direction. There are under the law 18 specific references to the Secretary having to prepare Regulations in order to operate the program. Although we have an Act, we do not have the Regulations, and until we get the Regulations, we will not know how the Act is really going to be implemented. These are the 18 areas in which the Secretary must perform his duties:

(1) Shall establish PSRO areas throughout the country by January 1, 1974.

(2) Shall designate conditionally those PSROs which qualify.

(3) Shall designate through regulation such other organizations which would qualify after 1976.

(4) Shall under regulations set out the criteria when and under what circumstances he can exercise his authority to waive any or all of the review, certification or other activities pursuant to the Social Security Act.

(5) Shall under regulations provide for notice to physicians and opportunity for a poll in any area as to whether the designated organization is representative of the physicians in the area.

(6) Shall promulgate regulations governing the manner in which PSROs will determine and publish the types and kinds of cases which will be reviewed.

(7) Shall promulgate regulations governing the authority of the PSRO to carry out its duties and functions.

(8) Shall promulgate regulations establishing the basis on which the PSRO may accept the findings of the review committees of hospitals. (A very important regulation; every hospital in the United States is anxiously looking forward to that regulation.)

(9) Shall promulgate regulations governing the form and contracts between the Secretary and the PSRO.

(10) Shall promulgate regulations on the manner in which the norms of care shall be developed for the type and extent of services and the type and extent of facilities needed.

(11) Shall promulgate regulation governing the manner in which PSROs will provide recertification dates for hospital admission.

(12) Shall promulgate regulations governing conditions for payment of claims, and conditions governing the hearing and review of claims for the beneficiary, practitioner and provider to appeal decisions.

(13) Shall promulgate regulations governing the manner in which providers and practitioners meet their obligations.

(14) Shall establish statewide Professional Standards Review Councils.

(15) Shall promulgate regulations governing the advisory committees to State Professional Standards Review Councils.

(16) Shall establish a National Professional Standards Review Council.

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Letter to the Editor

To the Editor:

I am simultaneously editing two books, one on the prevention and treatment of the medical problems of golfers and one on the prevention and treatment of the medical problems of swimmers. They are to be written by physicians for the golfer and swimmer. I would appreciate hearing from anyone wishing to contribute a chapter or who would be able to suggest someone to contribute a chapter for either or both of the books — golf and/or swimming. Any replies should be sent to the following address:

CLAUDE A. FRAZIER, M.D.
4-C Doctor's Park
Asheville, NC 28801

FROM THE SECRETARY'S NOTEBOOK — *Continued from Page 236*

their fine jobs, and Dr. Bostwick asked the House if this year's arrangement for the *House of Delegates' and Reference Committee meetings* (2 days) was preferable, and did they wish it continued. It was the majority *vote* to do the same next year.

Stenographic Record — A summary of the proceedings of the House of Delegates is being sent to the county presidents, and to the members of the House of Delegates. (The complete report is on file in the Association's office in Brunswick, where it is available to any member of the Association.)

The meeting was adjourned at 5:20 P.M. on Saturday, June 16, and at 5:10 P.M. on Sunday, June 17.

Patricia A. Bergeron
Secretary-Treasurer, M.M.A.

IMMUNIZATIONS AND OTHER MEDICAL CONSIDERATIONS FOR INTERNATIONAL TRAVEL
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indicate the condition for which the medication has been prescribed. This note should be carried with other travel documents in case a customs inspection should reveal the presence of these medications.

Our experience has been that the health information provided by the travel agencies is often quite inaccurate. Travel agencies have a particular tendency to confuse "requirements" with "regulations" and give their clients inaccurate advice. If any traveller has questions about the advice given by a travel agency, or if that advice differs from the general advice given in this article, they are advised to contact their physician or health department to get the correct answers. It would also be helpful if, once they find out the correct answers, they could let the travel agency know that their recommendations are inaccurate so they can be corrected.

MANY PSRO PROVISIONS INCUMBENT UPON REGULATIONS — *Continued from Page 241*

(17) Shall promulgate regulations on disclosure of information. And last, but not least
(18) He shall promulgate regulations providing for correlation of activities with intermediaries and carriers, and that very important regulation has not been drafted.

"Our understanding is that the Secretary is generally about a year away from concluding the establishment of these regulations. Some will come in a shorter length of time, for example, establishment of PSRO areas."

Finally, Mr. Tresnowski referred to the organization chart within HEW for establishment of PSRO supervision, emphasizing the complete control of the activity within the office of PSROs directly under Assistant Secretary Edwards, under the direction of William Bauer, M.D. He called attention to the planning going on through three equal task forces established by the Medical Services Administration, the Bureau of Health Insurance in SSA and the Health Services and Mental Health Administration.

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NEEDED: Cooperative Effort Between the Fields of Medicine and Education Which Includes Classroom Teaching for the Child With Learning Disabilities

JANET E. ORDWAY, M.D.*

The purpose of this paper is to suggest that through interdisciplinary efforts by the fields of medicine and education it is possible to teach learning disabled children successfully and maintain them at grade level within each school system. In order to make these interdisciplinary efforts succeed, these children can and must be identified in their kindergarten or first grade year, and placed in self-contained classes where all subjects are taught by the same specifically trained teacher within one room. They should be medicated if necessary and their parents counseled on how to cope with a learning disabled child. This plan has significance for the State of Maine because, as elsewhere, the learning disabled comprise 10-30% of the school population.

There are 265,291 school children in Maine.[1] This would mean, if one used a conservative estimate of 15% as Maryland did in the Chapman report to the Governor,[2] that there are at least 39,750 learning disabled children in Maine.

There are many terms used interchangeably to describe the learning disabled child — dyslexia, specific reading disability, brain-damaged child, visuo-motor perceptive disorder and others. The three terms that will be used in this paper are minimal brain dysfunction (MBD) used most frequently in medical literature and specific language disability (SLD) the term most often seen in educational journals, or the term which the writer prefers — learning disabilities. These terms will be used to describe symptomatology rather than etiology.

The learning disabled child usually manifests dysfunctions in the categories of motor activity and coordination, attention and perceptual function, interpersonal relationships, impulse control, and emotional control.[3] Academically he is often behind in reading, speech, and/or behavior even though his IQ is 100 or above. With the medical history, teachers comments, psychological testing, IQ and achievement testing and an additional test for specific perceptual handicaps, the diagnosis can be readily made.

Before the MBD or SLD child is of school age the diagnosis can frequently be made medically by listening to how the mother views the child at home, especially if she compares him with other children in the family. She will often describe him as "always on the move," impulsive, prone to temper tantrums, or "born with two left feet." Though he does not purposely break toys, the toys frequently seem to break when he plays with them. He is hard to switch from one activity to another. He doesn't seem to understand danger or respond to parents' discipline. The usual methods of control seem ineffective with this child.

Questioning further, one may elicit the following history. The child may have started to talk later than normal and continues overly long to

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use such babyish words as "frow" for "throw" or mispronunciations such as "aminal" or "emeny" for animal and enemy. His attention span is short and he goes from activity to activity staying with each one a very short time. Oral directions frequently have had to be repeated. In the case of the older school age child, one can often obtain a history of left-right confusion. The child puts shoes on the wrong feet or clothes on backwards. Handedness is sometimes established later than usual or there is ambidextrousness or the child is right handed and left eyed. Simple tasks such as tying shoes, setting tables, telling time or giving or taking instructions are often hard for the MBD child. The child is frequently enuretic.

The SLD or MBD child is many times first recognized after entering school. At this point, he is seen by the teacher as a problem because of his short attention span, inability to sit still, inability to easily follow directions, difficulty getting along with his peers and his feeling of being constantly picked upon. He is easily distracted by noise or visual stimuli, is a daydreamer, and has trouble stopping one school activity and switching to another. The teachers describe the SLD and MBD child as "immature," and not ready for the next grade. Often the child repeats kindergarten or first grade or is placed in a pre-first grade for those who are old enough for the first grade but not ready for reading. He has shown poor fine motor control, in writing, using scissors, and constructing simple projects. Letter and number reversals or inversions, mirror-reading or mirror-writing, and trouble sequencing lasts long after the others in the classroom have established left to right reading and writing patterns.

As the less severely handicapped children progress in their school careers, they are frequently described as underachievers and their teachers often say of them that they have the ability but "don't try." Often they are seen as behavior problems in the upper elementary grades. They are frequently disruptive in class, restless, provocative, argumentative, and turned off on learning. Some eventually become school dropouts and/or delinquents. They continue to read below grade level, have poor penmanship with letter and number and/or word reversals persisting. Many of these students often appear intelligent because their verbal vocabulary may be large and they can often talk intelligently and with great detail about some subject being discussed in the classroom.

Because these children must use their reading and language skills on the classroom-administered group, IQ tests and achievement tests, they often do poorly. The skills, of course, have been poorly learned because of their perceptual dysfunctions. They are also distracted easily in the group setting. A test such as the Wechsler Intelligence Scale for Children (WISC) individually administered often gives a truer value of the child's innate ability. One often finds an uneven performance with the child scoring high in some of the subtests and lower in others. Although, according to Wender[4] there is no characteristic pattern on these tests, usually reading, spelling, and punctuation skills are lower than mathematical skills.

It is important to identify these children as learning disabled as early as possible because they do not learn by the usual teaching methods or by the usual supplemental tutoring methods during their school careers.[5] Dr. Gilbert Schiffman,[6] former State Supervisor of Reading in Maryland, found that with special help for a given period of time 82% of the children identified in the first and second grades could maintain grade level. The percentage drops to 46% at the third grade if remediation and treatment starts at that time, and falls precipitously to 8% by the time the children start receiving help at the sixth grade level.

A small number of the MBD or SLD students can stay at grade level because of their higher than normal IQ or unusual determination. They correct their reversals quickly enough so that they lose only time and use other cover-up techniques. But they do read slowly and suffer the educational consequences of this slowness. These strongly motivated children could successfully utilize classroom multisensory teaching and then return to the regular classroom after a year or two of such special training. Others need special teaching for longer periods of time in these self-contained classrooms.

MBD children have difficulties in perception much like people who are color blind or tone deaf. They may have auditory perceptual defects, visual perceptual defects, problems of recall and/or audiovisual integration. They may have visual-motor incoordination. In order for language, reading and writing to successfully develop, there must be automatic association of the stimuli as they are carried over the visual-auditory-kinesthetic sensory channels or nerve pathways going to the cortex of the brain. These "input" sensory stimuli must then be integrated with past stimuli or concepts and stored for immediate or delayed recall. When they are recalled, they must then be sent out as appropriately responding "output" motor patterns. For the normal child, simple teaching of new steps for learning to take place is enough. For the SLD or MBD child who has various degrees of dysfunction in the above mentioned input-output system,

there must be reinforced learning patterns.[7,8] By knowing the childrens' perceptual problems, the teacher or tutor can stimulate the child to think more frequently in his weaker areas, thereby reinforcing the developing patterns and gradually building up the child's confidence in these areas. He must simultaneously develop his inner resources so he can encode and decode, or trace and retrace the steps himself so eventually he can confidently function alone.[9]

But because of the great variation found in individual children, such development of inner resources has been handled for the most part on an expensive one-to-one relationship between a specially trained tutor and the SLD child rather than in groups. Since this one-to-one technique has often been a strain on school budgets, many children in Maine get less than adequate educational help in the area of specific learning disabilities. Successful passage of L.D. 965‡ by the Maine legislature would require the State to enforce the present law which assures every child the right to quality education and would place a further strain on the budget if the same one-to-one tutoring continues to be the method of helping SLD or MBD child. New methods have gone beyond this successful but expensive method. It has now been demonstrated that SLD children can be taught in groups and maintained at grade level.[8] It is this writer's opinion that a total, daily, coordinated program is more effective and less expensive in the long run than either one-to-one tutoring or resource center teaching for part of the day.

Classroom teaching of the children, even though the children vary in the types and/or severity of their individual handicaps, has been done in Western United States in some of the public schools in Washington, Montana, California, Oregon and Texas.[9] And it has been possible to maintain the children at grade level. All children in the school are tested for specific language disability at the end of kindergarten year or at the beginning of the first grade year. Three tests are usually given and evaluated: Metropolitan and Pitner-Cunningham Tests and Pre-Reading Tests such as the Slingerland Screening Procedures. This would mean that the 10 to 30% of the children could be identified within a school or S.A.D. system in Maine and grouped in one classroom for each grade as needed. If there were not enough SLD or MBD children from the grade, those children who showed a maturational lag could be added to make the class of about 15 to 20 in size. The cost to the schools would be the cost of initially training a teacher, or teachers in a specialized month long training course and supervision and coordination of the program. The program could be started at the first grade level and yearly expanded to include the second, and third grades and so on as needed. At Menlo Park, California they have special classes for the first six grades in self-contained rooms in the elementary schools using the state selected textbooks which all the other children are using in their regular classes.[10] Of course, the teaching methods differ considerably for the SLD child as do the teaching aids but the teaching aids can be made by the classroom teacher so that they are not costly. The children in the SLD program learn by firmly establishing patterns along audio-visual-kinesthetic-motor channels so that they learn to write, encode, decode, read and conceptualize. Even though the children are taught in self-contained rooms they are within the regular school building and mix as much as possible with the other children in the cafeteria, on the playground, in gym and other programs and they are not aware that they are being educated in any "special" way.

In the Slingerland method[8] of classroom teaching (one of several effective methods used today) the specifically trained teacher has the children form letters or letter combinations by writing them with his arm in the air, name the letters, see and say the key words, say the sounds, write them and remember them by any rules or cues which will help the children establish firm pathways over which they can travel eventually alone.

The idea is to associate the weaker modality or modalities with the stronger modality so that learning takes place. For example, phonics helps those who have visual dysfunctions. Much repetition of what is heard and felt associated with visual cues helps those with auditory perceptual weakness. For those with weaknesses in both the visual and auditory areas, much effort must be spent on "overteaching" in all areas — visual, auditory and kinesthetic. Some children may move ahead more quickly than others because of variations in either intelligence or severity of dysfunctions; but because the children's handicaps are known by the teacher, she can reinforce or supplement as necessary. By breaking each step into small components and asking children individually to supply answers in what appears to be random selection, the teacher can keep all the children thinking each step along the way, while staying within the "shortened" attention span of these children.

Once the diagnosis of SLD or MBD is made, the parents are often relieved to find that the difficulty is within the child and not related to something they have or have not been doing at home while raising the child, or children. The

‡L.D. 965 was passed by the 106th Legislature.

same inability to pattern that has been seen at school is seen in problems of behavior at home. The child is often unable to associate cause and effect and has to be told over and over that certain behavior will result in certain consequences. He doesn't seem to learn as readily as the average child from his mistakes. Because the average methods of limit setting are ineffective, parents will try a variety of methods hoping that something will be effective. Many SLD children are hyperactive, and this aggravates rather than improves behavior, and frequently wears their parents' temper thin, so that it is often very hard in the best intentioned parent to handle a situation coolly and consistently. Sometimes in attempts to work out solutions to the behavior problems one parent will become permissive and the other will be strict. This only confuses and frustrates a child who needs consistent and repeated limits to establish firm patterns to help him develop self control.

About 70 to 80% of hyperactive SLD children will respond to medication. Usually they respond paradoxically to such stimulant drugs as dextroamphetamine or methylphenidate which decrease hyperactivity and distractibility, and increase attention span, persistence, and perhaps even the ability to learn and retain learning. Other drugs have been used like the tricyclic antidepressants and the phenothiazines, diphenylhydantoin and mysoline, but on the whole seem much less effective than methylphenidate which is the drug of choice. It is started in small doses, 2.5 mgm to 5 mgm per day, depending on the age and weight of the child and gradually increased by 5 mgm increments every 5 days until the optimal dose, usually somewhere between 10-40 mgms, is obtained. Usually the drug is given before breakfast and at noon. If there is a response, it will be seen in the first 2-3 weeks and be observed by all teachers, parents and physicians. The child is more integrated, less hyperactive, and his attention span is longer. He does not feel as picked upon and he is more available to suggestions. Because of these changes, he is easier to teach at school and easier to live with at home. He feels now that he can succeed even if only in small ways.

Hyperactivity in some children may be innate, but it is often increased in severity following increased psychological stress. If, for instance, the child is in a grade for his chronological age level but has academic skills of a much younger child, he cannot do his work, and becomes confused, restless and/or bored, and finally disruptively hyperactive. When tasks are given that are commensurate with the child's skills, hyperactivity decreases. Having a child repeat a grade is often not the solution to the academic or social problems as the language arts skills may still be lower than the repeated grade level.

The etiology of SLD or MBD is unclear. Probably because there may be several separate causes of MBD. The condition may be inherited,[3] it may be an environmental insult, or as Morrison and Stewart suggest it might be some sort of genetic-environmental-endocrinologic interaction.[13] Prematurity, complications of pregnancy and birth or early developmental difficulties are elicited in many histories but in many others there is a history of learning disabilities or behavioral problems in the family history. For reasons unknown, there are four to five times more MBD boys than girls. Wender[4] suggests that there may be chemical changes within the brain related to the metabolism of the monoamines which may be abnormal in the learning disabled child.

Before making the diagnosis of idiopathic MBD or SLD, one must first rule out a number of other diagnoses. Loss of hearing, peripheral visual problems, and children with emotional problems or subclinical epilepsy may produce an "idiopathic" MBD-like picture. The MBD child who is also either retarded or has gross neurological brain damage must be diagnosed and treated differently than the SLD or MBD children discussed in this paper. Any additional diagnoses resulting from such studies must necessarily lead to additional therapy.

Whatever the cause or causes of the learning disabilities, the educational needs of these children are the same. The previously mentioned multisensory approach of laying down basic learning patterns is needed.

Since the MBD or SLD syndrome can be greatly modified and improved by medical and educational efforts, the children must be identified as early as possible in their school careers because in an untreated or unrecognized form, MBD can lead to severe secondary emotional and social consequences. Though the signs and symptoms of the syndrome are varied, the children can be identified by medical history, teacher or school evaluation and tests for perceptual dysfunctions. Once the diagnosis has been made, special education using the specialized multisensory approach in self-contained classrooms, parent counseling, and in some instances, medication will help the children. Such a medico-educational team method would be a way for Maine to help these children maintain grade level with little additional cost to the public school system.

REFERENCES

1. Department of Educational and Cultural Services, Maine Educational Directory, 1972-73, Augusta, Maine.

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Special Article

A "Current Status" Report – second edition – concerning Maine students who have graduated from the University of Vermont College of Medicine under the aegis of the New England Higher Education Compact during the years 1961-1973.

GEORGE T. NILSON, M.P.H.*

BACKGROUND

Expansion of opportunities within New England for medical and dental education was the primary concept behind the New England Higher Education Compact. The first effort of the New England Board of Higher Education was to implement this idea. Arrangements were made during 1958-59 whereby certain states, i.e., Maine, Massachusetts, New Hampshire, and Rhode Island — would provide subsidy for such of their residents as were admitted for these studies to schools within the region. One condition for this subsidy was a guaranteed expansion of enrollment. It was on this point that those early arrangements failed, since the private colleges of medicine and dentistry were unable to meet this condition. Although a few students were enrolled at the private schools under this program in its first year, the Board discontinued this part of the activity and proceeded to arrange contracts between the four states and the College of Medicine at the University of Vermont where expansion of enrollment had taken place. These contracts were signed in 1960 and 1961. They provided that:

1. The University of Vermont would guarantee places for specified quotas of qualified students from each state, and would charge each enrolled student only the in-state tuition fee.
2. The contracting states would provide toward the cost of education for each enrolled student an annual payment of $2,500.00‡
3. These state payments would be processed through the New England Board of Higher Education, which would also certify the residential eligibility of all applying students, and render appropriate accounting.

The quota of Maine students began at fifteen (15) per academic year for all four classes in September 1961. The enrollment of Maine students by year and number is listed below.

Over the ensuing years, this quota has been increased and now stands at forty (40). Although the question of committed service (indenture) has been raised upon occasion, this has never been required of the participating students. This follow-up study was undertaken for the purpose of securing information as to the present whereabouts and future plans of these young physicians.

Year	*Number of Maine Graduates*
1962	4
1963	3
1964	3
1965	1
1966	3
1967	6
1968	4
1969	2
1970	3
1971	5
1972	7
1973	10

FINDINGS

Total number of University of Vermont College of Medicine graduates from the State of Maine assisted under the New England Regional Medical Student Program in the years 1962 through 1973 .....(51)

Number responding to follow-up questionnaire .....(41)

I. *Present Status*
- Military Service .....(5)
- Military-Internship .....(0)
- Military-Residency .....(4)
- Internship .....(8)
- Residency .....(13)
- In Practice .....(11)

II. *Future Plans*
- Private Practice .....(14)
- Institutional Practice .....(5)
- Other .....(3)
- Group .....(10)
- Private Practice-Group .....(9)

A. *Location*
- Maine .....(28)
- Other N.E. State .....(3)

*Maine TB and Health Association, 20 Willow Street, Augusta, Maine 04330.

‡This subsidy was increased to $5,000 in 1971.

Other state .....(6)
Other country .....(1)
Undecided .....(3)

B. *Type of Area*
Rural .....(8)
Semi-rural .....(15)
Suburban .....(8)
Urban .....(9)
Undecided .....(1)

C. *Type of Practice*
Family Practice .....(7)
Specialty .....(34)
Other .....(0)

1) *Specialty Preferences*
Anesthesiology .....(3)
ENT .....(0)
General Surgery .....(4)
Internal Medicine .....(5)
Orthopedic Surgery .....(2)
OB-GYN .....(6)
Ophthalmology .....(1)
Pathology .....(1)
Pediatrics .....(4)
Psychiatry .....(4)
Radiology .....(2)
Other sub-specialty .....(2)

III. *General Comments Made by the Respondents*

Without the help from the State of Maine to Vermont (UVM Medical School) I could not have gone to medical school. My state income tax has repaid my educational costs to the state many fold. Charity work has also repaid my community and state. I urge you to *subsidize* the med student when he *needs help* — while he is a student. This is the cheapest way that Maine can obtain MD's.

* * * * *

Although I am a proponent of a Maine Medical School (there are several well-staffed hospitals in the state which could supply clinical experience, and any one of the present "college towns" could easily adapt to the Basic Science Curriculum), I believe the present subsidy program is the only way in which to induce students with marginal finances to pursue a medical education. The program must expand to meet the demands of a growing population and increased sophistication of medical practice. It would be cheap at twice the price!

* * * * *

I am very indebted to the New England Medical student program allowing me as a resident of Maine to attend UVM. I'm strongly in favor of continuing this program even after establishing a medical school within our own boundaries, which I understand is in the process of formation. If Vermont can do so, shouldn't Maine! I only regret that I have not returned to Maine for practice. I summer in Maine and my family are voters. I will contact my "state" representative to vote favorably.

* * * * *

The UVM program gives Maine more medical graduates per dollar than any program we could create within the state. I feel more places at UVM should be obtained (if possible) rather than set up a medical school of our own. Whatever you do, don't give up the UVM medical program!

* * * * *

I begin work as a staff psychiatrist for the Counseling Center in Bangor, Nov. 1, 1973, and will be working primarily in Hancock County. I feel the agreement with Vermont is still essential. From what little I've read of plans for a Maine Medical School, i.e., 3-years (1 year basic sciences, 2 years apprenticeship with Maine physicians) I worry about Maine's compromising quality of care for numbers of doctors.

Also, those of us who leave the state contact more people to potentially come to Maine. For example, one of my colleagues here is starting at the counseling center the same time I am. Another colleague is definitely interested when he gets out of the service next year and at least 2 GIs are going to be looking us over after we get settled because of an active interest in Maine.

* * * * *

Formation of "regional centers" to the exclusion of small community hospitals is essential to providing good medical care for the following reasons:

a) 24-hour service for walk-in care of sick (non-emergency) patients until 9 or 10 at night, and emergency patients all the time.
b) Promotion of education in medicine fostered by larger groups of doctors through better communication, and house staff or student training.
c) Attract higher quality physicians in all areas.
d) Provide specialty care in all centers.

Maine seems to be promoting the "regional" concept better than many other states.

* * * * *

Without the program, I would have been unable to attend medical school. UVM is a top notch school which is established. Starting a

Maine Medical School would be too expensive, especially with Federal funds cut off.

* * * * *

Until you have a Maine Medical School, UVM is, in effect, our state university medical school. At only $5,000 per spot, the cost to the state is a bargain. The more you can buy, the better.

* * * * *

I am still considering the General Pediatrics in Maine once my obligations to the military are ended.

* * * * *

Perhaps some sort of newsletter put out by the Maine Medical Association or other appropriate group would be helpful in listing vacancies for medical practice in Maine. This would be especially helpful to those who are doing residencies geographically remote from Maine.

* * * * *

The more slots you are able to get in a UVM class the better. I believe this is the correct way at the present to secure medical college education for the State of Maine. UVM is able and willing to take many more students for the first two years from Maine and have them return for the clinical years to places like MMC. If this is not acceptable in some circles — as I know it isn't — the more individual places obtained — the better.

* * * * *

I agree that the NERMSP is an excellent substitution for a medical school in the State. I feel it is only a temporary solution to increasing the medical manpower of the state however, and when plans begin to establish a medical school in Maine utilizing its vast clinical resources at Portland and educational facilities at Orono, I shall give it wholehearted support and wish to be contacted.

* * * * *

There is no question in my mind that the small investment (relative to the costs of educating the same number of students in Maine) involved is the biggest bargain the State of Maine has ever had. So what if *all* the students *don't* return to Maine — *many do*. I support your efforts wholeheartedly. I strongly support the idea of a "combined" medical school for Maine and Vermont, with basic science (i.e., 1st 2 years) instruction in Vermont and clinical science experience (last 2 years) in some of Maine's excellent clinical facilities.

* * * * *

Maine sends the fewest students per capita (to med school) in the nation. The state would be ill advised to finance a state medical school (which it certainly can't afford). Money would be best spent to increase the number of students at UVM and the number and quality of internships and residencies available in Maine.

* * * * *

I completely support your efforts to increase Maine's quota in the UVM Medical School. For me, this program was the only way to get to go to medical school both from a financial aspect and from simply having a seat available in a medical school for me. The education we get costs Maine $5,000/yr., Maine could duplicate this only at a cost of $50,000/yr., as has been the results of University of Massachusetts and the University of Connecticut. Therefore, I believe Maine is getting a bargain and ought to increase the program.

* * * * *

I think that Maine has to consider the fact that physicians tend to stay to practice in areas where they did their residency training rather than where they went to med school — thus it would be more logical for Maine to develop more residencies and put pressure on those hospitals in Maine who offer internships/residencies (now only Maine Medical Center) to (1) upgrade their programs to attract more Maine applicants, and (2) to offer positions to those people — especially Maine residents who apply: I think efforts should be made to train medical students who grew up in rural areas and wish to return. Very few medical schools/residencies are available where students are exposed to rural patients who in fact are as distinctly different in medical problems and attitudes to medical care as center city people. Thus I would see advantage to a person who was from a rural area (in Maine) and who qualified for medical school to be urged to continue his training where he could get experience with rural populations. The University of Vermont is an excellent medical school for this. In terms of residencies I can only speak of psychiatry residencies of which there are only a few rural programs in the N.E. and even fewer which are good. It would be nice if some of these made an effort to accept people into these programs who had rural backgrounds and would most likely stay in the rural areas to practice. I.e., if you have qualified rural Mainers, don't make them go elsewhere for residency training because they will get settled there and you'll lose them.

* * * * *

I think this program is vital for many of Maine's would be physicians. In support of continued growth of the NEBHE-UVM plan I would like to point out that I think the statement ". . .,

it was not always possible to recruit a sufficient number of qualified Maine students to meet the quota guarantee set by the contract." Vol. 62, No. 4, page 78, Maine Medical Association, April 1971, is somewhat misleading for the following reasons:

a) the yearly statistics indicating the number of Maine graduates does not take into account the attrition rate due to low grades; illness; change in vocation; etc. This is a factor in determining the total number of recipients of state aid. Once a student leaves school, his or her place remains vacant unless filled by an occasional transfer student. This may account for the low 1965 output and for the low overall yield.

b) I think that there is more than an adequate number of qualified applicants to medical school from Maine. As indicated in your cover letter, 'over 100 young people . . .' applied to UVM in 1973. Apparently there has been a change since 1971. This should be emphasized if we are to increase our subsidized positions from 30 to 40 per year.

IV. *Summary*

Fifty-one Maine persons have been graduated from the UVM-College of Medicine under the aegis of the New England Regional Compact during the thirteen year period (1961 through 1973). Twenty-seven (27) are in practice and twenty-four are in a graduate training status. Of the twenty-seven, nine (9) or 33 1/3% are presently practicing in the State of Maine. Fifteen or approximately 55% are practicing away from Maine and three are apparently career officers in military service.

Of the twenty-four in training status, only two are at the Maine Medical Center — the others are scattered around the country. Twenty-six of the graduates now in training indicate a preference for Maine as a future practice location.

Opinions of the Author:

1. The State of Maine-UVM Medical Student Program proves to be increasingly viable when viewed against the experience of other states in developing tax-supported medical schools.
2. Although the Regional Medical Student Program was never intended to be a major solution for supplying Maine's physician manpower, it has the potential for doing just that as more Maine based residency training programs are developed. It is well recognized and sufficiently documented that the majority of physicians tend to locate in areas adjacent to where they received their hospital training.
3. In the interests of better coordination and more effective utilization of Maine's medical education resources, I recommend that the administration of the Maine Regional Medical Student Program be removed from the Maine Department of Education and Cultural Services and the New England Board of Higher Education and placed within the office of medical education in the University of Maine. These two agencies (NEBHE and Dept. of Education and Cultural Services) perform as administrative functionaries. Neither has a commitment to physician education or health care. It is expected that the University of Maine would be a creative and responsive manager of the Program.

ACKNOWLEDGMENT

I am indebted to David M. Tormey, M.D., Associate Dean of the University of Vermont, College of Medicine and the cooperation of his office and to Daniel F. Hanley, M.D., Executive Director, Maine Medical Association for his encouragement.

Physician Held Liable for Failing to Fill Out Health Insurance Form

A treating physician may be held liable for damages resulting from his neglecting to fill out an insurance form, a Delaware trial court ruled. The court held that the filling out of medical insurance forms is one of the duties owed a patient by his attending physician.

The physician had treated a patient for a kidney infection and anemia. When the patient applied for health insurance soon afterward, the kidney infection was listed on the application and a short form seeking information concerning the infection was sent to the physician.

The form was not completed by the physician for several months, although numerous requests had been made by the patient's husband and the insurance agent for him to do so. Because the form was not completed in time, the insurance policy was not issued, and the patient and her husband sought to recover damages for medical expenses they had incurred during the interval in which they had no medical insurance.

The court dismissed suits charging the physician with interfering with contractual relations and with fraud because it was not shown that he had intentionally failed to fill out the form. However, the court ruled that he could be held liable for negligently causing the losses sustained if a physician-patient relationship was proven in subsequent proceedings.--*Murphy v. Godwin*, 303 A.2d 668 (Del.Super.Ct., Feb. 15, 1973) — *The Citation*, September 1, 1973, Vol. 27, No. 10.

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Warnings: Use with caution in young children, because of variable response, and with extreme caution in patients with cirrhosis and other advanced hepatic disease or abnormal liver function tests, because of possible hepatic coma. Diphenoxylate HCl may potentiate the action of barbiturates, tranquilizers and alcohol. In theory, the concurrent use with monoamine oxidase inhibitors could precipitate hypertensive crisis.

Usage in pregnancy: Weigh the potential benefits against possible risks before using during pregnancy, lactation or in women of childbearing age. Diphenoxylate HCl and atropine are secreted in the breast milk of nursing mothers.

Precautions: Addiction (dependency) to diphenoxylate HCl is theoretically possible at high dosage. Do not exceed recommended dosages. Administer with caution to patients receiving addicting drugs or known to be addiction prone or having a history of drug abuse. The subtherapeutic amount of atropine is added to discourage deliberate overdosage; strictly observe contraindications, warnings and precautions for atropine; use with caution in children since signs of atropinism may occur even with the recommended dosage.

Adverse reactions: Atropine effects include dryness of skin and mucous membranes, flushing and urinary retention. Other side effects with Lomotil include nausea, sedation, vomiting, swelling of the gums, abdominal discomfort, respiratory depression, numbness of the extremities, headache, dizziness, depression, malaise, drowsiness, coma, lethargy, anorexia, restlessness, euphoria, pruritus, angioneurotic edema, giant urticaria and paralytic ileus.

Dosage and administration: ***Lomotil is contraindicated in children less than 2 years old.*** Use only Lomotil liquid for children 2 to 12 years old. For ages 2 to 5 years, 4 ml. (2 mg.) t.i.d.; 5 to 8 years, 4 ml. (2 mg.) q.i.d.; 8 to 12 years, 4 ml. (2 mg.) 5 times daily; adults, two tablets (5 mg.) t.i.d. to two tablets (5 mg.) q.i.d. or two regular teaspoonfuls (10 ml., 5 mg.) q.i.d. Maintenance dosage may be as low as one fourth of the initial dosage. Make downward dosage adjustment as soon as initial symptoms are controlled.

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Dosage forms: Tablets, 2.5 mg. of diphenoxylate HCl with 0.025 mg. of atropine sulfate. *Liquid,* 2.5 mg. of diphenoxylate HCl and 0.025 mg. of atropine sulfate per 5 ml. A plastic dropper calibrated in increments of ½ ml. (total capacity, 2 ml.) accompanies each 2-oz. bottle of Lomotil liquid.

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In Praise of Restraint

BERNARD L. MACKINNON, M.D.

We hear a great deal today about the need for honesty and openness. "Letting it all hang out," if not a universally revered goal, is certainly both a popular and esteemed practice. We hear very little about what we in Psychiatry call "ego strength," a term which refers, among other psychological functions, to our ability to select, measure, modify and control what we say and what we do. Ego strength has fallen on evil days. Many people seem to forget that man is a creature of reason as well as of emotion, that our rationality more than anything else differentiates us as a species. Pascal was right when he said, "the heart has reasons which the reason does not know," but he was an eminently reasonable man and would have been among the first to cry that when man discards his reason he discards his humanity. In fact, he also said, "Man is only a reed, the weakest thing in nature; but he is a thinking reed."

It is true, of course, that man is not fully man without sensitivity, without an awareness of and an ability to express his feelings. It is true also that one can be too much the rational being and over-select, over-modify, over-inhibit and control one's natural instincts and feelings, and that this too diminishes man and often gets him into psychological difficulties. One of the frequent tasks of psychotherapy is to help a person to get in touch with his real feelings, to face them and to accept them, and yes, to let them all hang out, no matter how base they may seem. But this is in therapy, not in life, and this is an important distinction. Some people need to learn to loosen the reins on their sexuality and their aggression not only inside the therapist's office, but outside, too. But control and selective inhibition are still necessary and the development of these abilities is an important part of the therapeutic task. The process of "letting it all hang out" is often a part of psychotherapy but only enroute to a recapturing of a more fully human state in which really letting go is rarely indicated, in which thought and feeling are melded, and impulses and primitive emotions are recognized for what they are and selectively expressed, modified and inhibited. In fact, most psychiatric difficulties come not so much from the inhibition of open expression of feelings as from fooling ourselves about the true nature of our feelings. Of course, the well balanced person is not often conscious of having to exert such control but it is there, nonetheless. And when it is not, the deficit should be recognized for what it is and not elevated to the status of a virtue.

Unfortunately, the fetish of brutal honesty and untrammeled emotionality has become an end in itself to a multitude of confused and misguided people (including some who are called "therapists"), who believe that such wallowing in the id is healthy and good and, somehow, through fullness of expression, will make them better, more complete, more fulfilled and, therefore, more human. It is human also to fear and to envy, to hate and to lust, to lie, to kill, to rape and to rob — but surely part of our task as human beings is to modify and control these impulses and these acts, and that it is partly by doing so that we grow towards the fullness of our humanity. People must have opportunities now and then to let go totally, to put control on the shelf for a while but, depending on the impulse being yielded to, the setting, and the degree of the negation of control, the result may be among other things, an orgy, a crime, an intuitive decision, or recreation, or therapy, but not a consistent behavioral goal in itself for a human being.

What I have said applies not only to our so-called baser emotions, but also to those which are more universally approved. Love is an in-word and an in-feeling, and so are "openness" and "community" and "sharing" and "reaching-out." These words express desirable human qualities and I do not wish to seem scournful of them, but I am turned off by the extremism of their application. What about "privacy" and "discretion" and "discrimination" and the sort of honesty which differentiates between degrees of love and the appropriateness of openness, and refuses to be bamboozled into manufacturing and expressing false feelings out of fear of being inhibited. Can love be indiscriminate? Can openness be total? And if they can, should they be? Again, our reason, our ego, if you will, is utilized here, and certainly not always consciously, to assess and select and appropriately to modify the expression of thought, feeling and behavior.

This is not meant to suggest that, even if it were possible, reason and emotion should always be in equilibrium. What a loss of unique and idiosyncratic human nature that would be. But some balance is necessary, a different sort of balancing act for each of us, and this accomplishment is the desirable human quality which gives man dignity and uniqueness and strength.

Continued on Page 254

Special Article

My Most Interesting Cases

EDWARD BLUMBERG, M.D.*

As an old physician in retirement one is inclined to settle accounts and to strike balances, also of the medical experience gathered in more than fifty years of medical practice. First question: which were the most interesting cases or the most striking observations?

Puerperal TETANUS is certainly one of the rarest and most surprising complications after deliveries and abortions, at least in this era of asepsis and antisepsis. I had the opportunity of observing such a case; I could clarify, how the entrance of the bacilli into the uterus had taken place.

It was the first phase of my medical activities, a general practice in Leipzig, Germany, with a lot of gynecology and obstetrics. Abortions were frequent, seldom spontaneous, mostly started by a professional abortionist or by the pregnant woman herself. The physician was called to treat a hemorrhage or to terminate an incomplete abortion, which was mostly done at the home of the patient. The intervention was, of course, legal, and there was no law that suspicious cases should be reported to the authorities.

The abortion of Mrs. N. belonged to this category. She had been pregnant for about three months, had expelled the fetus on the preceding day and called me because of a moderate hemorrhage. I removed the retained placenta without difficulties, using properly sterilized instruments and gloves. The postoperative course did not show complications until the tenth day, when Mrs. N. appeared at my office complaining about difficulties to open her mouth. On examination, she presented the ominous spasm of the jaw muscles, the trismus, which left no doubt about the diagnosis: tetanus. I did not allow the patient to return home, but I transferred her by ambulance to the Hospital of the University of Leipzig. She had her first convulsions during the trip to the hospital. There she was treated with injections of Magnesium Sulfate, the most efficient drug at that time, and was saved. After her discharge from the hospital, Mrs. N. confided in me and answered all my pertinent questions in detail. She conceded to have performed the abortion herself by an intrauterine irrigation with water from a vessel, in which SHE HAD WASHED AND PEELED POTATOES a short while ago.

This information solved the question how tetanus bacilli had entered the pregnant uterus and at the same time threw light upon the problem how the endemic outbreaks of tetanus in the lying-in hospitals of the 18th and early 19th centuries may have originated.

The well known political events in Germany forced me to a quick emigration to Bolivia in 1939. For the first two years, I worked as a physician of the Bolivian Government in the most remote, only half-civilized provinces among the native Indians, entirely dependent on myself. The intentions of the Government were excellent, but my equipment was despairingly insufficient. I fought against tropical diseases, against typhus, typhoid fever, smallpox, handicapped by the lack of basic remedies. What can you do, when during a smallpox epidemic the available vaccine turns out to be inefficient and it takes more than two weeks before you get another supply?

What did the Indians themselves do to defend themselves against disease? I had a wonderful opportunity to study the folkloric medicine of a certain region, as for most of the time my headquarters was the small Indian town of Charazani, (Prov. Munecas, today Saavedra). From this corner of the old Inca empire, now belonging partially to Bolivia, partially to Peru, two highly important plants have entered the pharmacopeia of modern medicine, the leaves of the Coca plant and the bark of the Cinchona tree. Both are used there as a household remedy for all kinds of illnesses. But I could learn more at Charazani. This region was the center of traditional Indian medicine, which has its roots in the era of the Incas and of the Spanish conquest. From here, migrant "curanderos" cross the South American Continent, offering their "professional" help everywhere. They are easy to recognize by a special uniform with a large bag of their medicinal herbs and are always ready to enhance the effect by a bit of black magic. I could keep up quite friendly relations with these "Callahuayas" (Indian name for medicine men). I was told that the herbs had to be picked under the full moon, but also that the Callahuaya by virtue of his magic power could endow any object (which the patient had to carry with him), with healing qual-

*Brooklyn, N.Y., formerly Pineland Hospital, Pownal, Maine

ities. I learned that dried Llama-fetuses were an all purpose drug; also that a frog put into the bed of a patient would absorb the disease (still more effective when the frog had been blinded). When there was a delivery, I saw a band of drummers around the house, making as much noise as possible to chase away the evil demons, etc. etc. Once I had a bad experience with a witch doctor. An Indian had played with dynamite and the explosion had destroyed his forearm, so that the bonestumps were exposed. While I sterilized the instruments for an operation, the man disappeared. He had gone to consult a witch doctor, who apparently dissuaded him from undergoing the operation. I saw him again only after two weeks, with gangrene of the arm, which shortly after led to his death. — I myself could not escape the glory of having magic power, as thermometer and stethoscope were considered as the most modern inventions in the field of magic medicine.

One case of my own practice appears worthwhile to relate. I was called to a man with all signs and symptoms of intestinal obstruction, due to a visible and palpable tumor of the size of an apple. I could not do very much for the man. Operation was out of the question and I had not even morphine available. So I gave him a strong dose of barbiturates hoping for nothing better than a quiet night. To my greatest surprise, the man himself came to see me early in the next day, grateful, beaming with delight and with something awful in a bag: a heap of worms (Ascaris), closely entangled, slowly moving, a true head of Medusa. Evidently the barbiturates had worked not only on the patient, but also on the parasites, which had lost their hold in the intestines and were expelled.

Another remarkable case from Bolivia (this time from the capital La Paz, where I practiced after passing my Bolivian License Examinations), was the improvised treatment by an Indian family of an obstetrical complication, eversion of the uterus after delivery. Apparently the family had felt the necessity of doing something before my arrival, possibly against hemorrhage, and so I found a dirty string wound around the everted and prolapsed uterus. Horror! My advice to transfer the woman to the hospital for immediate amputation (antibiotics were not yet on the market) was not accepted and so I could only remove the string, treat the affected area with a topical disinfectant and replace the organ. And . . . nothing happened, not even an elevation of temperature. I assume that the string had not come in contact with the inner surface of the uterus.

Another question which many old physicians have put to themselves at the end of their career: How many lives have you saved which would have perished without your presence or without your extraordinary effort. I can report only two cases as certain assets: One is the version from a transverse position which I performed on an Indian woman in the wilderness. She would have died without intervention, and no other physician could be reached at a distance of at least 500 miles.

The other case sounds so unlikely that almost every colleague, whom I told about it, expressed doubts about the diagnosis: sarcoma of the mesenterium. I would not have included this case into this paper, if I had not found a satisfactory explanation for a miraculous cure in a recent publication, from which I will quote below.

The patient was a man in his twenties, who had emigrated to Brazil some years ago and who had returned to die in his native country after his disease was found to be incurable. Twice a laparotomy and biopsy had been performed; the first time in Brazil, then in Hamburg. Both times the histological diagnosis had been the same: sarcoma of the mesenterium, and the abdomen was closed without further intervention. X-rays were tried without effect. When I saw the patient for the first time, the abdominal cavity was filled with an enormous amount of ascites, which I removed by paracentesis. This procedure had to be repeated every five or six days; each time more than two gallons were withdrawn. At that time, there was much experimentation with intramuscular injections of the patients' own blood, and one of the foremost cancer investigators, Blumenthal, suggested to use this method against malignancies. Another suggestion to enhance the effect of these injections was to sensitize the patient by preceding injections of sera of other origin. I used bovine and equine sera, easily available as diphtheria antitoxin. I tried it, as there was nothing to lose in this hopeless situation. The success was the most stunning of my medical career. In the 24 hours after the injection of his own blood, the patient emptied the whole amount of ascites by vias naturales, and there was never again any accumulation of fluid in the abdomen. The patient fully recovered (another serial of x-rays were applied). He remained without recurrency under my observation for 15 years, until 1939, when I saw him for the last time. Only a small hard fibrotic tumor remained palpable.

I had always assumed that the formation of antibodies was the cause of this sudden change, but only recently I read about experimental confirmation in the article mentioned above: William C. Wood and Donald Morton, Host Immune Response to a Common Cell Surface Antigen in Human Sarcoma, New England Journal of Medicine 285: 567, 1971. I quote two sentences: "Im-

munization of patients with sarcoma with their own irradiated sarcoma cells induced an increased immune response to the common serum antigen" and "Tests suggest the possibility of favorably altering the immune response of a patient with sarcoma to his tumor." — It is gratifying to get something explained after fifty years of waiting.

It may appear strange that I cannot add one case of my activities at Pineland. All institutions for mentally retarded patients are rich sources for rare syndromes, but I feel that they are more of interest for the geneticist than for the clinician.

There are two categories of "interesting cases." An example for the first category are cases as they are presented in clinicopathological exercises. They can be diagnosed only by having recourse to all modern ancillary services (x-rays, lab., etc.) and by the most careful evaluation of the results. I would compare the enjoyment in studying such cases with the solution of a chess problem. The cases in my report do not belong to this category. They have one thing in common: the surprise element, which makes them comparable to a mystery story. — Well, from time to time everybody reads a mystery story.

NEEDED: COOPERATIVE EFFORT BETWEEN THE FIELDS OF MEDICINE AND EDUCATION WHICH INCLUDES CLASSROOM TEACHING FOR THE CHILD WITH LEARNING DISABILITIES — *Continued from Page 246*

2. Chapman Report to the Governor and the General Assembly of Maryland, P.O. Box 402, Cockeysville, Maryland, Commission on Dyslexia, September 27, 1972.
3. Thompson, Lloyd J.: "Learning Disabilities: An Overview," American Journal of Psychiatry, 130:4, April 1973.
4. Wender, Paul H.: "Minimal Brain Dysfunction in Children," Wiley-Interscience (Division of John Wiley & Sons, Inc.), New York, 1971, 242 pp.
5. Eisenberg, Leon: "The Epidemiology of Reading Retardation and a Program for Preventive Intervention," The Disabled Reader, *edited by John Money*.
6. Schiffman, Gilbert: "Early Identification of Reading Disabilities," Bulletin of the Orton Society, Vol. XIV, 1964.
7. Gillingham, Anna and Stillman, Bessie: "*Remedial Training for Children with Specific Disability in Reading*, Spelling and Penmanship, Educators Publishing Service, Cambridge, Mass., 1970, eighth edition.
8. Slingerland, Beth H.: "A Multi-sensory Approach to Language Arts for Specific Language Disability Children," Educators Publishing Service, Inc., Cambridge, Mass. 1971, 239 pp.
9. Slingerland, Beth H.: Preventive Teaching Programs in the Classroom: A General Education Responsibility, Bulletin of the Orton Society, Volume XXII 1972, Towson, Maryland.
10. Gott, Joan, personal communication.
11. Conners, C. K., Rochschild, G., Eisenberg, L., et al.: Destro-amphetamine Sulfate in Children with Learning Disorders," Archives of General Psychiatry, 21:182-190, 1969.
12. Conners, C. K., Eisenberg, L.: "The Effects of Methylphenidate on Symptomatology and Learning in Disturbed Children," American Journal of Psychiatry, 120:458-464, 1963.
13. Morrison, James R. and Stewart, Mark A.: "The Psychiatric Status of the Legal Families of Adopted Hyperactive Children," Archives of General Psychiatry, June 1973, Vol. 28, No. 6, pp. 888-891.

ACKNOWLEDGMENT

To Dr. John Fahey, Dr. John Ordway, and Mrs. Anne LaBossiere, I wish to express deep appreciation for their considerable contributions to this paper.

IN PRAISE OF RESTRAINT — *Continued from Page 251*

I have been a member of training groups and sensitivity groups and many times, after a session, would know in my bones that I had participated in a ludicrous fraud. Much of what is purported to be honesty in these groups is the worst sort of phoniness and a dishonest negation of human values. It is undeniable that some people do benefit from such group experiences. They can be therapeutic, or in groups not set up for therapeutic purposes, they can enhance one's ability to communicate and to comprehend the nuances of interaction among individuals. But the influence of such groups is particularly noxious when their leaders fail to make the proper distinction between values to live by and temporary, artificial values which are sometimes successful in promoting a therapeutic process.

The fallacies of the encounter and sensitivity group movement have found expression in and have been fed by the fallacies rife in society at large. The current emphasis on open emotionality and the abhorence of discipline of feeling constitutes a shift towards barbarism — a diminution of our humanity not a fulfillment of it.

What we need and what we have always needed is not rational man and not feeling man, but man who acts on the basis of reason and emotion, judiciously mixed.

---

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Special Article

Summer Externship — 1973

JOHN W. GEORGITIS

This summer's externship was truly an enjoyable and enlightening experience. It has shown me at this point in my medical education what to expect from physicians and what they expect me to know. It has also shown me that the specialist must know the basic sciences better than the general practitioner; however, both practice good medicine. Every doctor practices in his own way and treats his patients accordingly. Patients understand this and it is one of the reasons he or she goes to this specific M.D. But, this does not mean that the doctor is the only health officer practicing good medicine. The community can by providing necessary ambulance and emergency care facilities. I have seen communities that have made many strides in this direction and I have seen others that have made no changes at all. I have also observed both the political and economic factors at work in the hospital and in individual practices. It is possible for a student like myself to help make improvements by asking very probing questions and perhaps by initiating special projects during my third and fourth year monthly rotations. Therefore I can help medically both the community and the doctor.

I have also found that both the patients and the staff in the hospital have taught me many things. Through these people, I was better able to understand the many complicated relationships found in the practice of medicine. I feel I am better equipped after this summer to deal with such people and the different situations that may arise. I am sure now that I wish to be a doctor, but I have not yet decided whether to be a primary care physician or a specialist. This summer's experience has also convinced me that Maine is the state in which I want to practice.

As for future educational opportunities, I hope to do a year and a half of monthly rotations with the physicians I worked with this summer. All of them were receptive to having me for a month as a fourth year student. With the discussion of a Maine Medical College, I could possibly be a pilot student to test the feasibility of sending students for their basic science training at another medical school and returning for their clinical training in Maine hospitals. However, to do this, hospitals and doctors must be willing to redefine some of their duties. I must be able to take a patient and be responsible for his care while in the hospital. This responsibility should include writing orders, assisting with operations, and making a diagnosis. Essentially I would be an acting-intern helping to admit patients, caring for them and following through with their care after they are discharged. Hopefully I will receive permission from the University of Vermont to pursue this course of study. I feel that in order to become a well-trained M.D. I must be able to take care of patients. This is what the Vermont Medical School does and the students and doctors involved find this approach to be very successful. I feel that such a program can be established in Maine and it will provide the necessary requirements for effective clinical rotations.

Finally, I wish to thank the members of the Maine Medical Association and the Regional Medical Program for allowing me to do this externship. I recommend that if possible the program should be continued. It was very rewarding for me at this stage in my education.

Lamoille House, Apt. 46, Fort Ethan Allen, Winooski, Vermont 05404

—News From Blue Cross and Blue Shield—

THE CONSUMER, BLUE SHIELD AND MEDICARE

Ned F. Parish, President of the National Association of Blue Shield Plans, (NABSP), made the following comments about consumerism and the relationship of Blue Shield with Medicine at the Annual Business Meeting of Blue Shield Plans.

A few years back, our business wasn't so complicated, and one could feel comfortable knowing that even if we made the wrong decision, people would give us credit for good motives. Our critics were few, because most people were pulling for us to make it.

Well, we made it. Blue Shield is the largest medical-surgical prepayment system in the world. We are no longer the underdog struggling for survival; we are the establishment. We have attracted a lot of critics. That can be helpful because it requires us to examine ourselves. Listening to other points of view builds perspective. Accepting them builds character.

One of the most forceful factors in the environment is consumerism. Consumerism is a barometer that measures the public's rising expectations.

It's tempting to assume either of two extreme positions regarding consumerism. One is to try to ignore it — the other is to give the store to anybody who wants it. Of course, neither of these is a lasting solution to anything.

One example of consumerism's effect on Blue Shield is the increasing pressure on Plans to change the composition of their Boards of Directors.

I find it difficult, however, to listen to someone like Commissioner Denenberg, who demands that the consumer be recognized and that his voice be heard by Blue Shield — even to the extent of advocating 100% consumer Boards of Directors.

Who does the Commissioner think we have been listening to these past thirty years?

How could we have reached our present position as the dominant organization in health care prepayment without the consumer?

The benefits, cost, and servicing of our bellwether national accounts — motors, steel, telephone, FEP, as well as much of our local business — are all the result of discussion and negotiations between medicine and hospitals, as the providers of care, labor and management, as direct purchasers of that care, and Blue Shield and Blue Cross as the implementing mechanism for each. To me, this represents the strongest kind of consumer input.

If Blue Shield is to respond to the public's rising expectations, both of us must be realistic about what can reasonably be expected. I suspect another expression is about to creep into our vocabulary — "consumer overpromise." I fear the consumer is going to be promised considerably more than business can deliver — something we saw happen in politics in the sixties. The result was disappointment and bitterness for millions of Americans who had expected great changes in both their country and their life styles.

If Blue Shield should leap headlong into public domination of Plan Boards, with the implied promise that things will suddenly be different, we would be misleading the public. Considerable unrest would follow, when rates continue to increase — as they will — and certain claims continue to be denied — as they will.

I want to emphasize again that we must not — we cannot afford to — weaken our relationships with the medical profession. On the contrary, we must continue to strengthen them. But Blue Shield and medicine are going to have to clarify their complementary roles.

Certain developments in the health care field, such as Foundations for Medical Care and Professional Standards Review Organizations, make it urgent that we move actively to keep those roles complementary rather than slipping toward the competitive. Hopefully, the medical profession will see that the truly useful role of the Foundation and the PSRO is one of responsibility for the quality and necessity of health-care. Blue Shield is properly responsible for claims processing, using guidelines developed in cooperation with the medical profession. Unless these

Continued on Page 259

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News, Notes and Announcements

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Through Examination

Abraham, George; Agana, Rudegelio A.; Ahmad, Ijaz; Ahmad, Nazir; Ahmad, Shireen; Aldea, Adrian; Alizadeh, Jamshid S.; Almazan, Ruben; Almendral, Doroteo M.; Alonso, Luis; Amin, Rohidas R.; Andalkar, Ratnakar R.; Antar, Robert E.; Antoon, Alia; Araneda, Luis O.; Askary, Nasser A.; Ayoub, Adel H.; Azimi, Farhad; Aziz, Kalim U.; Balteanu, Cornelia; Barbosa, A. Jerry; Beloin, Richard M.; Bertino, Frederic J.; Bhattacharjya, Ajoy; Bhimani, Gulam H.; Bollaci, Frederick A.; Brescia, Donald; Broadhead, Alan J.; Ceccacci, Luigi; Chandra, Suresh R.; Chang, Kuang H.; Chang, Po-Fu; Chaudhuri, Debi P.; Cho, Yong; Chrucky, Roman; Comentucci, Richard B.; Cordaro, Anthony F.; Dake, Peter D.; DeMartino, Joseph; Dobrovic, Slavo; Doraiswami, Subbiah; Dutta, Debabrata; Eamkeo, Chookiert; Eamkeo, Pranee; Eid-Khoury, Lily; Ellen, Ira J.; Essack, Joosub Y.; Fine, Kenneth C.; Fok, Shiu-Kwan; Foroutan-Yazdian, Manouchehr; Gallo, Victor A.; Gharai, Nader; Ghatak, Parimal K.; Glancz, George; Granmayeh, Masood; Grodin, Richard W.; Hathout, Ragaa M.; Hsu, Wen-Hsien; Inguagiato, Peter J.; Jaffe, Jonathan R.; Kamath, K. Prabhakar S.; Kanca, Ludmila; Kanca, Milan; Karunaratne, Esiriwickrema R.; Kaswan, Malcolm J.; Katz, Rozalia; Keomuan, Poonsri; Keszler, Kornelia; Khanna, Trilok S.; Khongrakul, Kitkasame; Kim, Kyoung J.; Kim, Taewoong; Kleinman, Lawrence M.; Ko, Taek Yong; Kohan, Silviu; Koka, Babu V.; Kurtz, Lewis M.; Laine, Saidel; Lasko, Keith A.; Lee, See Nam; London, Howard B.; Mahmood, Tariq; Mancheno, Mario A.; Marrero, Vito A.; Master, Herbert B.; McCleary, Wilfred D.; Mehandru, Sushil K.; Menon, Chandra S.; Miller, Albert A.; Moon, Sanggiu; Moreano, Walter F.; Nagia, Ali H.; Nissanka, Manage D. S.; O'Brien, Kevin M.; Pace, Salvatore A.; Pandey, Keshav D.; Parameshwaran, Sandirasegaram; Parameshwaran, Shantha V.; Park, Chang-Jo; Patel, Chandulal M.; Patel, Harendrababu, N.; Patel, Sureshchandra M.; Patkar, Jayant P.; Paverman, Jeffrey L.; Payumo, Carmelino C.; Piedrahita, Pablo; Politico, Jovencio; Qureshi, Sohail A.; Raghavan, Cheruvattath; Rao, Bhaskar N.; Rao, S. Vittal; Raval, Yagnesh R.; Reiss, David; Rocchio, Anthony G. B., Jr.; Rostas, Thomas J.; Sahandy, Parviz; Sahillioglu, Refik; Salib, Fikry B.; Sanchez, Rogelio; Santiago, Eufrocina A.; Sarakhun, Chalermpong; Satahusada, Rudolf M.; Seeliger, William R.; Shah, Devila P.; Shehata, Said A. H.; Shetty, Santosh K.; Shohadai, Seyed A.; Soto, Ana M.; Spindler, Joseph S.; Stamford, William P.; Striar, Jeffrey H.; Tchakarova, Bogdana; Vaccaro, Carmine A.; Varma, Surendra K.; Vedanthan, Pudupupakkam K.; Vladutiu, Adrian O.; Voleti, Choudary D.; Wadhwa, Vinod K.; Weber, Arthur H.; Weissman, Michael B.; Wilk, Ronald L.; and Zekri, Habib.

Through Reciprocity

Aselton, Carl K., Jr.; Barrett, John P.; Beals, Robert L.; Belanger, Charles F.; Belleau, Thomas; Bensen, Pamela P.; Brown, Lansing E.; Chai, Dou K.; Clark, William E., Jr.; Collins, A. Cowan; Cox, Paul M., Jr.; Curtis, James R.; David, Edward, Dela Cruz, Teodoro C., Jr.; Doyle, Robert P.; Elpern, David J.; Fakoory, Jacob J.; Goldberg, Morris M.; Giustra, Richard A.; Healey, James C. F.; Hedberg, Stephen E.; Herman, Richard M.; Ho, Che T.; Jerome, Alex W.; Jochimsen, Peter R.; Kneller, Leighton A.; Laperriere, Maurice; Malvesta, Robert J.; Martinak, Joseph F.; Maxwell, William H.; McCall, Ronald E.; McCarthy, Edward A., Jr.; McFarlane, Philip H.; McKee, Andrew D.; Miller, Halbert B.; Minihan, Patrick T.; Neuhauser, Edward B.; Pande, Sharad C.; Ratner, Jeffrey H.; Ray, Joges C.; Read, Francis A.; Rogers, Alan S.; Sewall, Kate; Skinner, David H.; Sodhi, Harbans S.; Soules, Virginia G.; Stoll, David A.; Stong, Frederick V.; Stram, Robert A.; Thurlow, William; Tse, Yat-Tung; Walker, Robert S.; Williams, Susan A.; Wilson, Donald W.; Wilson, Roger M.; Witwer, Timothy S.; and Zorick, Frank J.

County Society Notes

CUMBERLAND

The 379th meeting of the Cumberland County Medical Society was held September 20, 1973, at the Stage Coach Restaurant in South Portland, Maine. Sixty-eight members and guests were in attendance. All of the new physicians in the Portland area were invited as guests. Those attending were Drs. John Boothby, Brian Dorsk, Andrew Iverson, Jr., F. Stephen Larned, William H. Maxwell, Pierre E. Provost and Robert Doyle. The social hour was followed by a roast beef dinner. The President, Dr. Douglas R. Hill, called the meeting to order at 8:00 p.m. and introduced our guests. He also announced the election of Dr. Gisela K. Davidson to the Presidency of the Maine Thoracic Society. The reading of the minutes of the last meeting was omitted. The applications of Drs. Donald Leeber, Charles Thurber and Joseph Wilkis were given second readings and these gentlemen elected to membership.

First readings of applications of Drs. F. Stephen Larned, Elizabeth Serrage, Richard Herman, Donald Klopp, Francis Dyro, Timothy Carnes and Pierre Provost were presented. Dr. Klopp is a transfer from the Kennebec County Medical Society and Dr. Provost is a transfer from Middlesex West District Medical Society of Massachusetts.

Dr. Clement A. Hiebert presented a review of the technological advances in radio-paging systems. An in-hospital system is to be installed by the Maine Medical Center in the near future. Dr. Hiebert suggested that it will be possible to obtain wider coverage through a wide area transmitter. The hospital is not willing to assume this function and, if it is to become available, it must be carried by this Society or as a private business enterprise in this community. The initial investment for transmitting facilities carries a price of $15-$20,000.00. It is also felt that the leasing of individual receivers would average $20 per month. Much interest was expressed by members of the Society and we will conduct a mail survey to determine the number of individuals interested in a radio-paging system.

Dr. Robert E. McAfee, the delegate to the American Medical Association, presented a brief report on the recent meeting of the AMA. The main topic of concern was the increasing participation by third parties including hospitals in the practice of medicine. He indicated a weakening position of hospital staffs and physicians relative to the trustees of hospitals. A second point of concern was the lack of definition of the role of the private patient in the hospital. This is particularly evident in hospitals where it is necessary to staff teaching services. He also stated that there was little hope for change in the attitude of the IRS as regards physicians' fees under Phase IV.

Dr. Winton Briggs announced a program to be conducted by the medical assistants in the Portland area. This will be an edu-

cational series. It will be delivered on Thursday evenings for two hours concerning all aspects of the assistant's role in a medical office.

There being no further business to transact, the meeting was adjourned by Dr. Hill at 9:00 p.m.

ALFRED E. SWETT, M.D., *Secretary*

LINCOLN-SAGADAHOC

The regular monthly meeting of the Lincoln-Sagadahoc County Medical Society was held at the Ledges in Wiscasset, Maine on September 18, 1973. Dr. Peter A. Evans, the President, called the meeting to order at 8:45 p.m. The minutes of the last meeting were read and accepted as read.

Dr. Evans called for old business: Drs. George W. Bostwick and Richard C. Leck discussed the annual meeting in Kennebunkport. Dr. Anthony J. Horstman spoke on PSRO as anathema and felt that its opponents were not given a fair hearing at Kennebunkport.

The Secretary then read a letter from Dr. Richard Evans asking to be transferred to active membership. A letter from Dr. Mary J. Tracy was read, resigning as delegate because of retirement from practice in the area. Dr. Evans appointed Dr. Elihu York to serve in her stead.

Dr. Evans read a letter from the President of the Maine Medical Association asking for a county consensus of fees. Since the letter was written 29th June, the secretary was instructed to ask Dr. Paul A. Fichtner what current information is desired; the Society moved to postpone further discussion to a future meeting.

Dr. Bostwick read two requests from the Maine Medical Association. One was for any changes in membership categories of members. The other was for 1974 dues. After discussion, Dr. Charles E. Burden moved that County dues be raised from $30 to $35 annually; the motion was seconded by many and passed without opposition.

Dr. Horstman brought to the attention of the Society the fact that the M.M.A. has officially endorsed AMA Medicredit proposals in Congress.

The meeting was then turned over to Dr. Louis Bachrach, who told of Medicare support of fertility measures and then introduced Dr. Ronald A. Bettle of Brunswick, who spoke on his trip to Africa.

GEORGE W. BOSTWICK, M.D., *Secretary*

ANDROSCOGGIN

The meeting of the Androscoggin County Medical Society was held at Steckino's Restaurant, Thursday, September 20, 1973. The meeting was called to order at 8:00 p.m. by the President, Dr. Gilbert R. Grimes.

The minutes of the previous meeting were read and accepted.

Reports of the following standing committees were made: Upon recommendation of the Credentials Committee, the application of Dr. Pamela P. Bensen, Emergency Medicine Department, St. Mary's General Hospital to the A.C.M.S. was moved, duly seconded and voted to membership. Dr. Paul M. Beegel, reporting on representation to the Tri-County Health Planning Committee, indicated that Mr. Massden's report to that Agency, following his attendance at the Androscoggin County Medical Society meeting of April 19, 1973, did not fully express the opinion of this body in regards the "Feasibility Report of combined services of the Emergency, Obstetrics, Gynecology, Pediatrics and Pathology Departments of St. Mary's General Hospital and the Central Maine General Hospital by the C.M.G.H.-St. Mary's G.H. Liaison Committee." It would appear from Dr. Beegel's report that the Tri-County Health Planning Agency is striving to justify the mandate given it by its parent governmental agency and that delivery of medical care in the Tri-county area must suffer the growing pains of this pledging consumer group. The report of the Diabetes Committee to respectfully refrain from participation in Diabetes Week was accepted, and following motion and seconded, it was voted to refrain from participation in the Statewide Diabetes Week for 1973-74. Reports were presented by members of the House of Delegates re business conducted at the Maine Medical Association meeting in June. Dr. Donald Anderson presented his report of the Executive Committee meetings as Councilor for Seventh District.

The following physicians were nominated and recommended for special membership in the Maine Medical Association: Honorary Membership — Drs. Merrill S. F. Greene and Linwood A. Sweatt; Affiliate Membership — Drs. Ralph Zanca and Gilbert A. Clapperton.

A request from the President of the Maine Medical Association to provide information relative to individual fee schedules was presented. Any member wishing to provide this information to the M.M.A. in order to assist in establishing a Statewide uniform fee schedule may forward same to M.M.A., or to the Secretary of the A.C.M.A. for forwarding to the M.M.A.

It was brought to the attention of this group that increasing governmental pressure and regulations pertaining to care of the chronically ill in nursing homes, etc. has resulted in a drastic reluctance on the part of many physicians to participate in these Governmental-sponsored programs.

Dr. Grimes presented official complaints relative to timely completion of Death Certificates by one or more members of this Society. It was moved, duly seconded and voted that the Ethics Committee investigate the complaints and institute disciplinary action if indicated.

There being no further business, the meeting was adjourned at 9:35 p.m.

RICHARD M. SWENGEL, M.D., *Secretary*

OXFORD

A regular meeting of the Oxford County Medical Society was held at Bethel, Maine on October 3, 1973.

Dr. H. S. Sodhi was elected to membership in the Society.

The following officers were elected for 1974:

President: Dr. Alfred Oestrich, Rumford

Vice-President: Dr. Stephen B. Dewing, Waterford

Secretary-Treasurer: Dr. John B. Makin, Jr., Rumford

Delegate to the M.M.A. House of Delegates: Dr. David L. Philips, Rumford. Alternate: Dr. Warren C. Hazelton, South Paris

Councillors: Drs. Eugene Gorayeb, Rumford, Harry L. Harper, South Paris and Dexter E. Elsemore, Dixfield

STEPHEN B. DEWING, M.D., *Secretary*

THE CONSUMER, BLUE SHIELD AND MEDICARE

Continued from Page 256

roles are clearly drawn, and unless the traditional underwriting and marketing functions of carriers are recognized, some conflict is inevitable.

NABSP is moving to avoid this conflict by continuing to seek the understanding, support and cooperation of medicine. We work closely with the staff of the American Medical Association in many areas, and we have continuing liaison with the American Association of Foundations for Medical Care. But communication at the national level does not take the place of strong liaison at the Plan level. Operation decisions are made there.

Letter to the Editor

To the Editor:

August 1973

Apoligitica

For many months I have been solely tempted to write a letter, or statement to this journal regarding personal deeply held and felt views that bear on medical practice. Several factors, other than the usual ones of available time and willingness to make the effort, deterred me. Who would read my views? Are they relevant and important enough to ask others to take time out of their lives to read them? Won't I be ridiculed for retaining an old value system? Won't I offend and turn off some of my peers and colleagues by preaching to them? All these considerations pertain, but obviously I have answered them, one way or another, to my own satisfaction.

A statement of personal philosophy of medical practice in a free society.

Over the last several years as a Medical Oncologist, I have had at least the average physicians' experience in observing physical and emotional suffering in people, social and economic disruption in the families of people with cancer, and personal frustration with the current state of Medical Technology in the care of cancer patients. Recently there has been added to these problems the matters of government and political influence in medicine and the implied threats of a multitude of quasi-official committees intruding themselves into the medical scene with ever increasing force.

I look about me and see a medical profession deeply troubled, largely disorganized (from many points of view), unsure of its role and rapidly losing its prerogatives. A multiplicity of others, without qualification or personal responsibility, are suddenly "patient advocates" and are gaining statutory powers in the areas of medical care. A good example but by no means an isolated one, is the American Hospital Association's recent "Bill of Rights" much of which deals with the relationship of physicians to patients. I do not impugn the motives of "the new advocates." I do believe they overstate their responsibilities and area of competence. Physician and particularly organized medicine, have not taken advantage of newer economic and administrative methods and non-medical technological changes to maintain and improve the health status of our society. There has been gross failure by physicians to recognize that for a medical care system to be effective in caring for whole nations of people, an enormous spectrum of non-medical technology must be utilized and that it is currently available. On the other hand, non-physicians tend to view and treat medical care like a manipulative commodity. My view is that a patient's record or even a patient's blood may be manipulated and computerized; the whole person should not be, and if our society remains free, will not be. Unfortunately, physicians over-awed by the seeming solidarity of criticism heaped upon them, unable to individually resolve the new problems and without meaningful collective leadership, are rapidly acquiescing to demands that they abandon much of what western medicine has always implied. It is for these reasons I state my own views of medical practice and principle.

Propositions and corollaries: I begin this section with a series of seemingly self-evident statements.

1. No person or thing lives forever on planet earth — the death or "passing on" of all of us is inevitable. This is intellectually but not emotionally accepted in our society, which means in practice, it is not accepted.

2. It follows, therefore, that living forever on earth, or attempting to cause someone to live forever, is not a reasonable goal for our society or for "medicine." Neither physicians nor governments should promise everlasting life or even health to their constituencies.

3. The natural course of events for all biologic (and other) systems is for eventual malfunction (disease), gradual decline, and death. In this context "medicine" is truly an interventionist art and science, intervening in this natural process of decline as long as it is reasonable to do so. In a free society, it is absolutely essential that intervention or non-intervention be a joint decision between the individual person and his physician only, and that "reasonableness" be defined within this relationship.

4. What can a person expect from medicine and his physician? Above all he can expect fidelity — he can expect the honest application of medical knowledge on his behalf, as long as he and his physician mutually understand and agree upon the application of that technology. But he can also expect more. When, for whatever reason, the application of technology is no longer useful or acceptable, particularly in serious disease states, he can expect the continued care and interest of his physician. From the physicians' perspective, the goals and methods of therapy might change, but his relationship to persons under his professional care, *cannot*. For example, in my own situation, this means that I have as much responsibility for caring for a person when he is beyond medical technology, as when he is going into remission on active therapy for his cancer.

In the current public context, to achieve these perspectives there are at least 3 aspects of medical practice that physicians should find "non-negotiable" within or outside of the profession. These should be publicly stated in the very near future and loud enough to be heard.

(1) A patient may freely choose the physician responsible for his care. Conversely, a physician may agree or not agree to accept responsibility in the care of an individual. There should be clear understanding between all parties on this point.

(2) Physicians (and their patients) must be free to choose the method of therapy within the structure of accepted medical and scientific knowledge.

(3) A physician's fundamental responsibility is to the person for whom he accepts responsibility. Although physicians are aware of the legitimate concerns of hospital committees, insurance carriers, government and "society" etc., his prime responsibility must be the person and the needs of that person called "a patient."

I am aware that endless discussion could evolve over any or all of the above statements. It is my sincere hope that *some* meaningful discussion and action might be precipitated among my colleagues by these notes.

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The Natural History of Coronary Artery Disease*

JOE R. WISE, JR., M.D.**

The development of surgical techniques to revascularize the myocardium has stimulated a more precise definition and understanding of the natural history of coronary artery disease. Previous studies have dealt with the natural history of this condition but often the information available was of a general nature, somewhat sketchy and anecdotal and, prior to coronary arteriography, rather too imprecise to use as a standard of reference against which we could judge the results of surgical treatments.

It is clear that coronary artery disease is common, accounting for some six hundred thousand deaths in the United States per year. The Mayo Clinic study of the population of Rochester, Minnesota and epidemiologic information from England and Wales showed that coronary artery disease was the cause of death in 40% of the adult males.[1,2] A World Health Organization study of six countries in which coronary artery disease was most prevalent (USA, Canada, Scotland, England, Wales, Australia, Denmark) revealed that coronary artery disease was the cause of death in 25% of men between the ages of 35 and 44 years, and one-third of those in the 45-54 age group.[3]

The cause of the coronary artery disease, or coronary arteriosclerosis has not been defined, nor is it clear why the coronary arteries seem to be selectively involved nor why coronary artery disease is more severe in men than in women. We do know that atheromatous streaks are present within the coronary arteries of newborn infants and that even at this age these deposits are more extensive in male infants.[4] We know from studies of Korean War dead that coronary artery disease is already established in young adulthood. We have learned from serial coronary arteriography that coronary artery lesions progress, sometimes within a period of a few months. Certain risk factors such as high blood pressure, high serum cholesterol, diabetes and cigarette smoking seem to accelerate the development or the progression of coronary artery disease.

It seems that the coronary artery disease is more common presently than in previous years. Although William Heberden described angina pectoris thoroughly in 1768, and, although it was discussed in the first issue of the *New England Journal of Medicine* in 1912 and the *Journal of the American Medical Association* in the same year, despite this, in seven hundred patients that Paul Dudley White saw during his internship in 1912 to 1913 only seven had angina pectoris. In three of the patients, the angina was thought to be due to syphylitic aortitis, and in one patient, severe rheumatic heart disease with aortic regurgitation.[5]

What do we know about the prognosis or natural history of coronary artery disease? It would be more appropriate, of course, to discuss the prognosis based on the cause of the disease rather than the results of it. But, since the cause is not known, we must use a clinical evaluation. It is unfortunate that we often do not know that a patient has coronary artery disease until symptoms develop, by which time the disease is far advanced. The clinical spectrum of symptomatic coronary artery disease can be divided generally into three broad groups — stable angina pectoris, unstable angina pectoris and established myocardial infarction. Each of these groups

*Presented in part before the 1973 Scientific Session of the Maine Heart Association.

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can be considered separately since the prognosis or natural history in each group is somewhat different.

Myocardial Infarction

Data from the Framingham studies suggest that as many as 20% of myocardial infarctions may be silent or asymptomatic and detected only by subsequent electrocardiograms.[6] Following myocardial infarction, approximately one-half of the fatalities occur prior to the time of arrival at the hospital.[7] Most of these early deaths are young people whose prognosis would be anticipated to be good if they survived the initial few hours.[8-9] After arriving at the hospital, the mortality is near 20%. Norris et al have devised a prognostic index of patients with myocardial infarction which shows that the hospital mortality following infarction is effected by the patient's age, the electrographic site and extent of the infarction, the blood pressure on arrival at the hospital, the heart size, the presence or absence of heart failure and the history of previous myocardial ischemia.[10] Coronary care units have reduced the mortality in the hospital by approximately one-half. In a representative study in which consecutive patients were assigned randomly to a coronary care unit bed or a general hospital bed, the mortality of those patients in the coronary care unit beds was one-half that of those in the general hospital.[11] The risk of death continues after the patient leaves the hospital following myocardial infarction. The mortality following first infarction is about 5% per year increasing after the second infarction.[6] Norris's figures again indicate increased risk in older patients following severe infarction, patients with increased heart size, pulmonary vascular congestion or a history of previous myocardial ischemia.[10] Angina pectoris persists in approximately 50% of patients following myocardial infarction.

In summary, following myocardial infarction, 20% of the patients die suddenly prior to reaching the hospital. Of the 80 patients who reach the hospital, 20 die in the hospital and 60 live to return home. Of these 60 patients, 30 remain asymptomatic with a mortality of 5% per year. The remaining 30 patients have persistent angina pectoris.

Angina Pectoris

The natural history of angina pectoris until recently has not been so well defined. The largest series of conservatively treated patients with angina pectoris was reported by Block in 1952. He followed 6,882 patients for 5 to 23 years.[12] In these patients, the average mortality was approximately 9% per year. White reported similar mortalities of 10 to 11% per year in 500 patients followed 25 years.[13] Framingham population studies of 303 patients with angina pectoris had a mortality of 5% per year which was the same as those patients in the posthospital phase of myocardial infarction. In this latter study, men with angina pectoris had a 25% risk of myocardial infarction within 5 years following the onset of symptoms.[6]

Since, in these studies cited above, no coronary angiography was performed these groups of patients with angina pectoris are rather heterogeneous. While most patients with angina pectoris have severe coronary artery disease, all do not. It is of interest in this regard that approximately one-third of one group of patients referred to the Cleveland Clinic for coronary arteriography had normal coronary arteries.[14] Thus, the mortality reflected by these clinically selected patients may not be entirely accurate.

Ross and his group at Johns Hopkins therefore devised a system of scoring the severity and extent of coronary artery disease based on the coronary arteriogram.[15] The average mortality for 224 patients with angina pectoris was 4% per year, but, within this large group, the mortality varied from 1% to 7% per year depending on the extent and severity of their disease! The prognosis in these patients was more accurately predicted by the coronary angiogram than by the usual clinical criteria. Sones and his group at the Cleveland Clinic have extended these observations.[16] They have followed 590 patients with angina pectoris studied by coronary arteriography before the availability of coronary artery by-pass graft surgery. The mortality for this group of patients was 6 to 7%, but again within the group, the annual mortality varied from less than 1% for lesions of the right coronary artery to 10% for patients who had involvement of three coronary arteries. It appears, therefore, that, while angina pectoris usually indicates the presence of coronary artery disease, the prognosis is determined by the extent and the severity of this disease.

Unstable Angina Pectoris

Unstable angina pectoris, or preinfarctional angina, is an intermediate syndrome between stable angina pectoris and established myocardial infarction. This condition probably represents a situation in which the coronary blood flow is barely adequate at rest, whereas angina pectoris represents myocardial ischemia occurring only with the increased oxygen demand of emotional stress and exercise. Unstable angina pectoris often precedes myocardial infarction. Patients with angina pectoris often report change in the pattern of their angina prior to infarction.[17] This intermediate syndrome is characterized by the increased frequency and severity of angina which seems to be produced more easily, often chest pain at rest, frequently transient S-T-T wave changes during the chest pain and minor serum enzyme changes. Coronary angiography invariable shows significant coronary artery disease.

In a group of 100 patients with unstable angina studied at the Massachusetts General Hospital,

only six had myocardial infarction while confined to the hospital. However, following discharge, the mortality in the first year was 22%![18] A group of 140 similar patients with unstable angina pectoris followed for ten years was reported by Gazes and his group in South Carolina.[19] His patients could be divided into several groups based on the electrocardiographic changes. They were able to select a high risk group characterized by the occurrence of previous angina pectoris, persistent or recurrent chest pain during hospitalization and S-T-T wave changes which occurred with the chest pain. In the entire group of patients with unstable angina, there was 18% mortality in the first year; 20% of these patients had myocardial infarction within 8 months. In the high risk group, there was a 43% mortality during the first year; 35% of these patients having myocardial infarction within three months.

Noncoronary Artery Disease

There is a group of patients, often women, with apparently typical angina pectoris often with positive exercise test, who have normal coronary arteriograms. In these patients, the prognosis appears to be quite good; the majority improving and becoming asymptomatic.[20] It is of note however, that at least three of these patients have had myocardial infarction, and, in at least one patient, the coronary arteries and myocardium were normal at the time of post mortem examination.[20,21]

In summary then, the lot of the patient with coronary artery disease is not a happy one. Patients with angina pectoris can expect a mortality of 1 to 10% per year depending, not on the severity of the symptoms, but on the extent and severity of their coronary artery disease. Patients with unstable angina pectoris appear to have a mortality of 20 to 40% per year. Following myocardial infarction, 20% of patients die suddenly. Of the 80% who reach the hospital, an additional 20 will die before discharge. Sixty percent of these patients following myocardial infarction survive to go home and within this group one-half will have persistent angina pectoris.

Coronary artery disease is a major problem. It is a disease with a bad prognosis and every effort should be made to find ways to prevent it. In the interim, patients with coronary artery disease should be identified as soon as possible, their individual prognosis defined as clearly as can be, and the application of potentially corrective measures with an attrition rate less than that of the natural history of the disease should be explored.

References

1. Spiekerman, R. E.; Brandenburg, J. T.; Achor, R. W. P.; and Edwards, J. E.: The Spectrum of Coronary Artery Disease in a Community of 30,000. Circulation 25: 57, 1962.
2. Peel, A. A. F.; Semple, T. in "Textbook of Coronary Care" page 152, Charles Press, Philadelphia, 1972.
3. Oliver, M. F. in "Modern Trends in Cardiology" 2: 46, Butterworths, London, 1969.
4. Dock, W.: Why are Men's Coronary Arteries so Sclerotic? J.A.M.A. 170: 152, 1959.
5. White, P. D. in "Hearts, The Long Follow-Up," page 175, W. B. Saunders Co., Philadelphia, 1967.
6. Kannel, W. B.; Feinlieb, M.: Natural History of Angina Pectoris in the Framingham Study. Amer. J. Cardiol. 29: 154, 1972.
7. Fulton, M.; Julian, D. G.; Oliver, M. F.: Sudden Death and Myocardial Infarction. Circulation 39/40 Suppl. IV: 182, 1969.
8. Yater, W. M.; Welsh, P. P.; Stapleton, J. F.; Clark, M. L.: Comparison of Clinical and Pathological Aspects of Coronary Artery Disease in Men of Various Age Groups. Ann. Intern Med. 34: 352, 1951.
9. McNamee, B. T.; Robinson, T. J.; Adgey, A. A. J.; Scott, M. E.; Geddes, J. S.; Pantridge, J. F.: Long Term Prognosis Following Ventricular Fibrillation in Acute Ischemic Heart Disease. Brit. Med. J. 2: 204, 1970.
10. Norris, R. M.; Caughery, D. E.; Defming, L. W.; Mercer, C. J.; Scott, P. J.: Coronary Prognostic Index for Predicting Survival After Recovery From Acute Myocardial Infarction. Lancet 2: 485, 1970.
11. Hofvendahl, S.: Influence of Treatment in a Coronary Care Unit on Prognosis in Acute Myocardial Infarction, ACTA Med. Scan, Suppl. 519, 1971.
12. Block, W. J.; Crumpacher, E. L.; Dry, T. J. et al: Prognosis of Angina Pectoris, Observations in 6882 Cases. J.A.M.A. 150: 259-264, 1952.
13. White, P. D.; Bland, E. F.; Miskall, E. W.: Prognosis of Angina Pectoris. J.A.M.A. 123: 801-804, 1943.
14. Proudfit, W. L.; Shirey, E. K.; Sones, F. M.: Distribution of Arterial Lesions Demonstrated by Selective Cine Coronary Arteriography. Circulation 36: 54-62, 1967.
15. Gottlieb, C.; Friesinger, G. C.; Page, E. E.; Ross, R. S.: Prognostic Significance of Coronary Arteriography, Trans. Assoc. Amer. Physicians 83: 78, 1970.
16. Bruschke, A. V. G.; Proudfit, W. L.; Sones, F. M.: Progress Study of 590 Consecutive Nonsurgical Cases of Coronary Disease Followed 5-9 Years. Circulation 47: 1147-1153, 1973.
17. Solomon, H. A.; Edwards, A. L.; Killip, T.: Prodromata in Acute Myocardial Infarction. Circulation 40: 463-471, 1969.
18. Krauss, K. R.; Hutter, A. M.; DeSanctis, R. W.: Acute Coronary Insufficiency. Course and Follow-Up. Arch Int. Med. 129: 808-813, 1972.
19. Gazes, P. C.; Mobley, E. M.; Faris, H. M.; Duncan, R. C.; Humphreys, G. B.: Preinfarctional Angina — A Prospective Study — Ten Year Follow-Up. Circulation 48: 331-337, 1973.
20. BeMiller, C. R.; Pepine, C. J.; Rogers, A. K.: Long Term Observations in Patients with Angina and Normal Coronary Arteries. Circulation 47: 36, 1973.
21. Kimbris, D.; Segal, B. L.; Munir, M.; Katz, M.; Likoff, W.: Myocardial Infarction in Patients with Normal Patent Coronary Arteries as Visualized by Cine Angiography. Amer. J. Cardio. 29: 725, 1972.

Erythema Nodosum

OTIS FIELD JILLSON, M.D.*

Each year I see only a few patients with erythema nodosum, but to care for such patients requires a wide range of knowledge involving chiefly its etiology, differential diagnosis, and its course.

ETIOLOGY: There are two broad etiologic concepts: 1) a disease *sui generis* (idiopathic), and 2) a disease produced by microbial agents (viral, bacterial, fungal, Chlamydozoaceae), drugs, and such diverse diseases as sarcoidosis, ulcerative colitis, leprosy, leukemia, and collagen diseases.

Tuberculosis: On the tenth to the fourteenth day of the primary tuberculous infection, the tuberculin skin test becomes positive. It is at this time, at the onset of the "delayed" tuberculin type of sensitivity, that erythema nodosum rarely may appear. This occurs three times as often in women. When erythema nodosum occurs during childhood, tuberculosis of the primary type of *short* duration is the cause until proven otherwise.

In 1965, this tenet detected an epidemic of tuberculosis in a Michigan nursery school. A five-year-old girl was brought to the office of Dr. Frank J. Malje with erythema nodosum. Although erythema nodosum is considered a rare symptom of tuberculosis, because of her age, Dr. Malje did a tuberculin patch test. This was positive, as was the confirmatory chest x-ray. Several other children were then found to have tuberculosis before the carrier, a woman employee of the nursery, was discovered, and the epidemic was terminated.[1]

Coccidioidomycosis: Erythema nodosum also occurs under the same circumstances in coccidioidomycosis, i.e., at the time the coccidioidin skin test is becoming positive. Here the incidence of erythema nodosum is much higher — 30% in white women, and 2% in Negroes and Filipinos.[2] Many years ago in New Hampshire, I made this diagnosis based on the history of travel through the arid Southwest and the finding of tender nodules on the leg.

Streptococcic Infection: Certainly in adults, this is by far the most common infectious cause of erythema nodosum. Favour recovered B-hemolytic streptococci from the pharynx in 20 of 48 cases in which cultures were made one or more times, and once from the antrum and twice from tooth sockets.[3] (The focus of strep infection may also be in the skin, lymphatic vessels, and draining lymph nodes.) Other than the history and laboratory findings, an elevated antistreptolysin titer provides additional evidence of a streptococcic infection. Peak ASO reaction generally occurs five to six weeks after infection with Lancefield Group A streptococci.[4] In contrast to chronic infections such as tuberculosis and coccidioidomycosis, in which erythema nodosum is a single episode, here the disease may be recurrent with each new streptococcic infection.

Other Infectious Disease: Other infections associated with erythema nodosum are histoplasmosis,[5] psittacosis,[6] lymphogranuloma venereum,[7] gonorrhea,[8] and cat scratch disease.[9]

Sarcoidosis: Erythema nodosum may usher in this disease. Besides being more common in females, the race determines its incidence. "For example, the association of erythema nodosum with the onset of sarcoidosis is very common among young women in Sweden and England. It is also frequently encountered among women born in Puerto Rico who have migrated to New York City. In contrast, erythema nodosum ushers in the disease less frequently among Negroes who constitute the predominant group of persons affected by sarcoidosis in this country. It is apparently not seen at all among patients with sarcoidosis in Japan."[10]

Drugs: Sulfonamides, salicylates, iodides, and bromides are the most common drugs precipitating this skin disease. Sulfathiazole provokes erythema nodosum in 10% of patients with lymphogranuloma venereum so treated.[7] Many modern drugs including penicillin, Dilantin,® chlorothiazides, and contraceptive pills have also been implicated.[11]

CLINICAL MANIFESTATIONS: The primary lesions are nodules located bilaterally over the extensor legs and less often over the buttocks, elbows, knees, ankles, and rarely on the neck and face. The most acute, violent, and widespread example of erythema nodosum I have ever seen which involved the face was in a patient with Behcet's Syndrome. This has been a recurrent erythema nodosum.

The lesions appear in *crops*, the total number of active lesions being less than a dozen, each reaching its full size (1-5 cm.) within a day or two. The lesions may become confluent. Although ulceration has been described, it is not a feature of this disease, and its presence should place the diagnosis of erythema nodosum in doubt.[3] Initially, the *nodules are tender* and firm, but they involute in about a week or longer, undergoing the same color changes as a contusion. Because of the appearance of new lesions, the skin disease may persist for weeks to months. There may be associated erythema multiforme and up to 4+ pitting edema of the lower extremities. The sex ratio is three females to one male.[12] It is com-

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moner in the young, but age of onset depends somewhat on the etiology.

Systemic: Malaise and fever are associated findings, and 70% of patients have joint involvement. This may consist of arthralgia or acute arthritis with redness, heat, swelling, and pain on motion. The joints involved most often are the ankles and knees, although arthralgia of the wrists and fingers is not unusual. The arthritis may precede the onset of erythema nodosum by *weeks up to three months.* Cough may be an annoying symptom because of pulmonary involvement.[3]

PATHOLOGY: For the details of the histopathology, refer to Lever's textbook.[13] The salient histopathologic findings are:

1) A scattered irregular infiltrate among the fat cells of the subcutis, consisting of neutrophiles and lymphocytes in the early lesions, and of foreign body type giant cells and epithelioid cells in older lesions. There is no caseation.

2) The blood vessels, especially the veins, may show invasion of the walls by the inflammatory infiltrate, with marked endothelial proliferation.

LABORATORY FINDINGS: This varies with the etiology. Usually there is a mild anemia and slight leukocytosis. However, the latter is extremely variable, being from 12,000 to 25,000, or even less than 5,000 if there is an underlying virus infection. Elevated sedimentation rate and slight albuminuria are other nonspecific laboratory findings.[3] Roentgenogram of the chest should be done because approximately 10% of patients with idiopathic erythema nodosum will have enlarged hilar or bronchial lymph nodes which are symmetric and bilateral.[14] Favour and Sosman described a *perihilar clear zone* separating the sharply outlined shadows of the bronchial nodes from the mediastinal shadow of the heart and great vessels. Although this sign is not pathognomonic of erythema nodosum, it is a presumptive diagnostic finding.[15]

DIAGNOSIS: The finding of tender, bilateral, asymmetric nodules undergoing the same color changes as a bruise located over the shins is diagnostic of erythema nodosum.

DIFFERENTIAL DIAGNOSIS: In *erythema induratum,* the nodules ulcerate and are located also over the calves. The tuberculin test is not only positive, but there is also extreme hypersensitivity as demonstrated by intense, even bullous, reactions to the usual skin testing procedures.[2] This I believe to be a *sine qua non* for this diagnosis.

Although in *primary idiopathic thrombophlebitis* the lesions are tender, they are *linear,* appearing in crops wherever the superficial and medium-sized veins are located on the legs. This disease keeps reappearing for years and years.

Relapsing febrile nodular nonsuppurative panniculitis is characterized by recurrent episodes of fever associated with deep nodular lesions of the subcutaneous tissues, especially of the thighs and buttocks. Entire fat lobules are involved in the subcutaneous fat with inflammation. Healing results in depression of the surface skin due to local loss of subcutaneous fat.[16]

Included in the differential diagnosis are *cold panniculitis,*[17] *nodular vasculitis,* and *subacute nodular migratory panniculitis.* Initially, in the latter disease, one or more nodules appear unilaterally, spread peripherally while healing in the center. Later the disease may become bilateral and often symmetric. There is rapid resolution of the lesions following treatment with 3-10 drops of saturated solution of potassium iodide t.i.d.[18] Finally, I have seen two patients with *autoerythrocyte sensitization.* This occurs almost exclusively in young females with hysterical and masochistic character traits. The presenting lesions are painful ecchymoses, some self-inflicted, some from the slightest trauma. They have a marked sensitivity reaction to intradermal and subcutaneous injection of red blood cells or stroma.[19]

With hilar lymphadenopathy, *tuberculosis, sarcoidosis, lymphoma,* and *Hodgkin's disease* must be ruled out. In erythema nodosum, the lymph nodes attain maximum size at once and thereby exclude a diagnosis of Hodgkin's disease or other lymphomas. The enlarged lymph nodes of erythema nodosum regress slowly, disappearing within a year and often by the sixth month — too rapid for sarcoidosis. Bilateral, symmetric hilar lymphadenopathy is not characteristic of the primary complex of tuberculosis.[14]

COURSE: The course is variable. Crops of new lesions may appear for weeks to many months. Favour states that "usually acute attacks last one to five weeks and subside without residual signs or symptoms. Approximately one patient in ten, however, has an attack that smolders on for months to a year or more."[3]

TREATMENT: Rest and elevation of the legs is the essential treatment for the idiopathic type. Otherwise, the treatment is that of the underlying disease, such as using specific anti-infectious drugs, and avoidance of drugs when causative. Internal steroids were necessary and rapidly effective in the patient with Behcet's disease and concomitant erythema nodosum. Corticosteroid treatment is sometimes necessary in the treatment of the idiopathic type also.

PROGNOSIS: The appearance of erythema nodosum is a good prognostic sign in general, and especially in tuberculosis, coccidioidomycosis, and sarcoidosis. The glaring exception to this is the erythema nodosum type of lepra reaction. This occurs in lepromatous leprosy and is not beneficial to the patient. Treatment is difficult and relapses are frequent.[20]

Continued on Page 272

Relapse of Felty's Syndrome Following Splenectomy

ALAN W. BOONE, M.D. and STANLEY J. EVANS, M.D.*

After nearly half a century since Felty[1] first described the syndrome of rheumatoid arthritis, splenomegaly and leukopenia, a number of basic questions still remain unanswered. While splenectomy is held by most to be beneficial, there are some questions regarding the timing of the operation and its contraindications. Moore,[2] O'Neill,[3] and Sandusky[4] have described high rates of remission after splenectomy and few late relapses, while others such as Barnes[5] are more pessimistic about lasting good results, reporting continued leukopenia, infections and deaths. Spontaneous remissions, both without splenectomy and in relapse after splenectomy[6] make any judgment of therapy difficult. This uncertainty reflects the incomplete knowledge of the basic pathologic mechanisms underlying the leukopenia. The finding of leukocyte-specific antinuclear factors in this syndrome[7] and the results of leukocyte kinetic studies[8] point to an aberration of immune defenses and accelerated destruction of neutrophils. Practically nothing has been written regarding the management of the patient who relapses severely following splenectomy. We think that the following case history may offer help in the management of a difficult clinical situation.

CASE REPORT

J. D. is a 61-year-old white male who first became ill with arthritis involving the neck in 1962. This was followed by involvement of the hips and thereafter neck, shoulders, hands and wrists. A diagnosis of rheumatoid arthritis was made. Because of severe hip involvement with pain and limitation of motion, hip prostheses were performed in 1964 and 1966.

Hematologic complications were first noted in April 1970, when he was admitted to hospital with pain in his neck and shoulders. Examination revealed an acutely and chronically ill man with marked deformity and limitation of motion of the neck, shoulders, wrists and hips. There were no subcutaneous nodules and no pigmentation. The spleen was palpable 3cm below the left costal margin. The liver was not palpable. Examination of the blood gave the following results: hematocrit 41 percent, leukocytes 1,620/mm$_3$ with 50 segmented neutrophils, 1 nonsegmented neutrophil, 45 lymphocytes, 2 monocytes and 2 eosinophils. The platelet count was 200,000/mm$_3$. Latex screening was strongly positive. Roentgenograms of the joints showed changes consistent with rheumatoid arthritis. The bone marrow showed a slight increase in plasma cells and lymphocytes. The patient was treated symptomatically with salicylates and physiotherapy.

Six months later he developed balanitis with severe phimosis and was hospitalized. Blood examination on this admission revealed the hematocrit to be 36 percent, leukocytes 1,200/mm$_3$ with 10 segmented neutrophils, 80 lymphocytes and 10 monocytes, platelets 125,000/mm$_3$. The patient improved on penicillin therapy and was discharged after thirteen days.

Thirty days later he presented with nausea and vomiting. Examination revealed the spleen 9cm below the left costal margin. The leukocyte count was 800/mm$_3$ with 6 segmented neutrophils, 1 nonsegmented neutrophil, 1 eosinophil, 79 lymphocytes and 13 monocytes. The platelet count was 125,000/mm$_3$. The bone marrow was hypercellular with adequate iron stores and a slight lymphocytosis. Splenectomy was performed, the 1,050g spleen containing typical fibrotic and congestive changes. The postoperative course was uneventful and the leukocyte count promptly improved to 4,800/mm$_3$ with 56 percent neutrophils, by the fourth postoperative day. The leukocyte count at the time of discharge was 5,500/mm$_3$.

His course during the following fifteen months included only two minor infectious episodes. After this interval, he was again admitted to hospital complaining of cough, chest pain, fever and chills. There were rales at the right lung base and a chest x-ray revealed changes consistent with pneumonitis. The peripheral blood revealed an hematocrit of 48 percent, leukocytes 4,600/mm$_3$ with 14 percent neutrophils. Sputum cultures grew Pseudomonas sensitive to gentamicin, with which he was treated, followed by prompt improvement.

Three months following this he was again hospitalized with pneumonia due to Pseudomonas. The leukocyte count was 2,400 with no neutrophils observed. The bone marrow revealed abundant granulocytes together with increased lymphocytes. The patient received gentamicin and cephalothin. Prednisone 40mg daily and azathioprine 100mg daily were also given and were followed by improvement in clinical state and hematologic values. At the time of discharge the leukocyte count was 3,900/mm$_3$ with 39 percent neutrophils, 1 eosinophil, 39 lymphocytes and 21 monocytes.

The patient was followed on a weekly basis and maintained on prednisone and azathioprine. On this regimen, he remained infection-free with a leukocyte count of 4,000/mm$_3$ and neutrophils 40 percent or greater (Fig. 1). Three months after initiation of immunosuppressive therapy, the prednisone was tapered and discontinued and the azathioprine was discontinued two months later. The patient has had subsequent minor infections only and neutropenia has not progressed.

DISCUSSION

Although the initial hematologic response of this patient to splenectomy was less than complete, the clinical improvement was striking. Moore[2] has suggested that a lymphocytosis of the bone marrow may be a contraindication to splenectomy. Although this finding may have made the relapse more predictable, the operation apparently afforded the patient more than a year without major infections.

The ineffectiveness of corticosteroid therapy as an alternative to splenectomy is generally agreed upon.[3-6] Yet, the dramatic and sudden improvement of this patient at the time of severe clinical relapse, by its timing, is more likely due to prednisone than to azathioprine. It is difficult to judge from the

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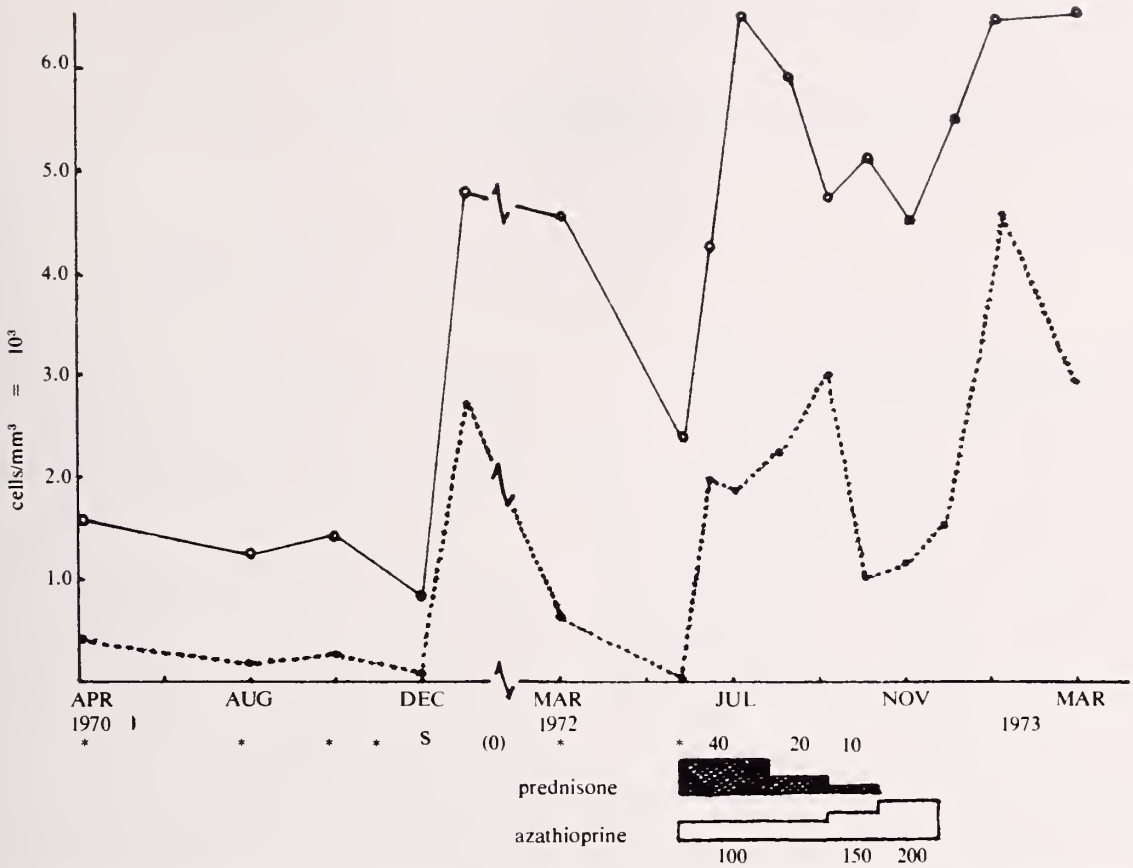

Fig. 1. Leukocyte count (solid line) and neutrophil count (broken line) dramatically improve after splenectomy (S) and after institution of immunosuppressive drugs. Infections requiring hospitalization (*) also become less frequent.

data whether the sustained partial hematologic improvement and complete clinical recovery were due to corticosteroids, to the purine antimetabolite, or to a spontaneous remission. The patient's stable course after both drugs were discontinued favors the latter possibility. The extreme rarity of severe, post-splenectomy relapse and the attendant clinical urgency for therapy make a controlled drug trial very difficult.

This unusual case brings up at least two points worthy of consideration in the management of Felty's syndrome. Lymphocytosis of the marrow may not be an absolute contraindication to splenectomy. Steroid therapy, while generally not helpful before splenectomy, could be important in a relapse following the operation, until the patient may enter an unmaintained remission.

Synopsis

A patient with Felty's syndrome attained a partial clinical and hematologic remission following splenectomy. Relapse in the form of agranulocytosis and Pseudomonas pneumonia 18 months later responded promptly to corticosteroids, azathioprine and antibiotics. Clinical well-being and partial hematologic remission have persisted after immunosuppressive drugs were withdrawn.

References

1. Felty, A. R.: Chronic arthritis in the adult associated with splenomegaly and leukopenia. Bull Hopkins Hosp 35:16-20, 1924.
2. Moore, R. A.; Brunner, C. M.; Sandusky, W. R.; Leavell, B. S.: Felty's Syndrome: long term follow-up after splenectomy. Ann Intern Med 75:381-385, 1971.
3. O'Neill, J. A.; Scott, H. W., Jr.; Billings, F. T.; Foster, J. H.: The role of splenectomy in Felty's syndrome. Ann Surg 167:81-84, 1968.
4. Sandusky, W. R.; Rudolph, L. E.; Leavell, B. S.: Splenectomy for control of neutropenia in Felty's syndrome. Ann Surg 167:744-751, 1968.
5. Barnes, C. G.; Turnbull, A. L.; Vernon-Roberts, B.: Felty's syndrome. A clinical and pathological survey of 21 patients and their response to treatment. Ann Rheum Dis 30:359-374, 1971.
6. Ruderman, M.; Miller, L. M.; Pinels, R. S.: Clinical and serologic observations on 27 patients with Felty's syndrome. Arthritis Rheum 11:377-384, 1968.
7. Faber, V.; Preben, E.: Leukocyte-specific anti-nuclear factors in patients with Felty's syndrome, rheumatoid arthritis, systemic lupus erythematous and other disease. Acta Med Scand 179:257-266, 1966.
8. Srodes, C. R.; Hyde, F.; Chervenick, P.A.; Boggs, B. R.: Neutrophil kinetics in Felty's syndrome. Abs 78 Am Soc Hematology, 15th annual meeting Dec., 1972.

Additional Observations on the Nevoid Basal Cell Epithelioma Syndrome

A Case Report

THOMAS L. WATT, M.D.* and FRANKLIN E. BRAGG, M.D.**

The Nevoid Basal Cell Epithelioma Syndrome[1] (or Basal Cell Nevus Syndrome) is an autosomal dominant hereditary trait consisting of the central triad of multiple nevoid basal cell epitheliomas, multiple jaw cysts, and multiple skeletal defects in addition to other variably expressed abnormalities. Its importance medically lies in the fact that early recognition of the syndrome can lead to early treatment of the cutaneous tumors which to the uninitiated might appear as innocuous moles, seborrheic keratoses, or comedones but are in fact nests of basal cells which can relentlessly progress into destruction and mutilating skin cancers.

The case to be reported calls to attention many of the regularly observed features of the syndrome, adds to the list of already reported skeletal defects, and relates our experience with the use of 5-FU.

Case Report

The patient, R. E., is a 16-year-old male who first received medical attention in 1958 and 1959 when he had a surgical repair of a cleft lip and cleft palate. In 1965, he had a "dentigerous cyst" removed from his mandible, followed in 1971-72 by the removal of several more such cysts from both his mandible and maxilla, both right and left. The product of a full term, spontaneous delivery, the patient is 72" in height with an armspan of 78". His lower jaw has protruded since birth and still does despite several orthodontic procedures. In addition to the typical lesions of acne vulgaris, multiple pigmented papules are seen over the face, eyelids, shoulders and back. On the palms and soles, there are numerous non-keratotic "pits." There are no cardiac murmurs. Neurological exam is normal. X-ray of the jaw and numerous pathology reports show typical "dentigerous cysts" but an x-ray skeletal survey fails to reveal any other skeletal abnormalities except a bifid left third rib. Blood tests revealed an alkaline phosphatase of 117 and 123 (normal up to 110), calcium 9.6 and 9.9, phosphorus 4.4 and 4.4 and albumin 5.5 and 5.5. Biopsy of several of the skin lesions revealed typical "basal cell epitheliomas" on pathological section. Repeated inquiries into the family history and examination of both parents failed to uncover similar abnormalities. No test was made for responsiveness to parathormone.

During November and December of 1972, 5% fluorouracil Solution (Efudex®) was applied topically to selected lesions, first over the left clavicle and then on the eyelids. The solution was applied with and without occlusion. Significant erythema developed on all areas. However, the chest lesion did not totally slough off after six weeks' application and the patient discontinued application to the eyelids after three weeks because of discomfort and feared potential injury to the eyes. Subsequently, nine lesions were removed via curettage and electrofulguration with good cosmetic results. Although no previously excised lesions have recurred, new lesions have continued to develop.

Discussion

The case reported exhibits the characteristic triad of multiple jaw cysts, multiple nevoid basal cell epitheliomas, and multiple skeletal abnormalities.

JAW CYSTS commonly are the presenting complaint for individuals with the syndrome. The cysts, by nature, are persistent, progressive, recurrent, and troublesome. Generally, the jaw cysts begin appearing in the first or second decade as painful swellings of the jaw which tend to displace teeth. Over the years, the cysts become bilateral and tend to involve the mandible more commonly than the maxilla. The cysts may be uni- or multilocular and may or may not involve teeth. X-ray studies readily demonstrate the cysts as radiolucent masses. The cysts are classified dentigerous or better as odontogenic keratocysts.[2,3] Histological examination of the typical cyst reveals a flattened lining composed of keratinizing squamous epithelium which encloses degenerating keratinous debris. The cysts in turn are surrounded by a dense fibrous capsule.

Occasionally, ameloblastic proliferation has been noted and in at least one case metastasizing fibrosarcoma developed.[4] The presence of the fibrous capsule is a source of difficulty for the oral surgeon.

Cutaneous findings are essential and diagnostic features of the syndrome. NEVOID BASAL CELL EPITHELIOMAS also generally begin developing during the first or second decade as grouped, small, skin colored or pigmented papules. The tumors can involve all skin areas but most commonly are located on eyelids, nose, nasolabial folds, and upper and lower lips or mid-facial areas. The lesions may remain quiescent for years, but their ultimate biologic potential is that of basal cell epitheliomas with accelerated growth, invasion, and ulceration. In fact, Taylor et al[5] cited two cases where direct invasion to the brain and brain and lung metastases occurred. It is for this reason that the designation of Nevoid Basal Cell Epithelioma Syndrome is

Continued on Page 269

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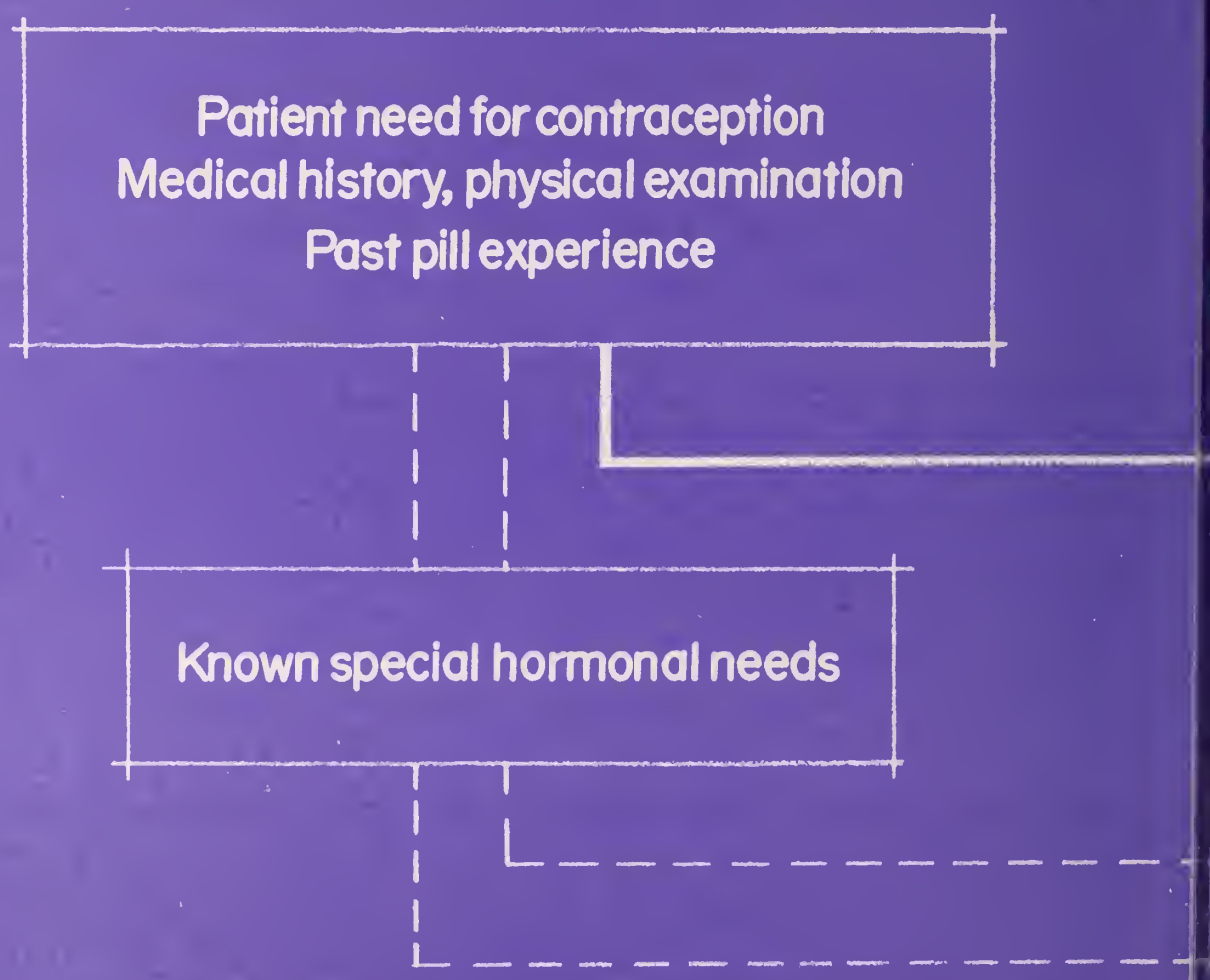

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sed primarily on animal studies.

Note: Oral contraceptives are complex medications. As with all medications they should be prescribed with discriminating care, and only after reference to full prescribing information. For brief summary of prescribing information, please see next page.

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Special note—Oral contraceptives have been marketed in the United States since 1960. Reported pregnancy rates vary from product to product. The effectiveness of the sequential products appears to be somewhat lower than that of the combination products. Both types provide almost completely effective contraception.

An increased risk of thromboembolic disease associated with the use of hormonal contraceptives has now been shown in studies conducted in both Great Britain and the United States. Other risks, such as those of elevated blood pressure, liver disease and reduced tolerance to carbohydrates, have not been quantitated with precision.

Long-term administration of both natural and synthetic estrogens in subprimate animal species in multiples of the human dose increases the frequency of some animal carcinomas. These data cannot be transposed directly to man. The possible carcinogenicity due to the estrogens can be neither affirmed nor refuted at this time. Close clinical surveillance of all women taking oral contraceptives must be continued.

Indication—Ovulen and Demulen are indicated for oral contraception.

Contraindications—Patients with thrombophlebitis, thromboembolic disorders, cerebral apoplexy or a past history of these conditions, markedly impaired liver function, known or suspected carcinoma of the breast, known or suspected estrogen-dependent neoplasia and undiagnosed abnormal genital bleeding.

Warnings—The physician should be alert to the earliest manifestations of thrombotic disorders (thrombophlebitis, cerebrovascular disorders, pulmonary embolism and retinal thrombosis). Should any of these occur or be suspected the drug should be discontinued immediately.

Retrospective studies of morbidity and mortality conducted in Great Britain and studies of morbidity in the United States have shown a statistically significant association between thrombophlebitis, pulmonary embolism, and cerebral thrombosis and embolism and the use of oral contraceptives. There have been three principal studies in Britain[1-3] leading to this conclusion, and one[4] in the United States. The estimate of the relative risk of thromboembolism in the study by Vessey and Doll[3] was about sevenfold, while Sartwell and associates[4] in the United States found a relative risk of 4.4, meaning that the users are several times as likely to undergo thromboembolic disease without evident cause as nonusers. The American study also indicated that the risk did not persist after discontinuation of administration and that it was not enhanced by long-continued administration. The American study was not designed to evaluate a difference between products. However, the study suggested that there might be an increased risk of thromboembolic disease in users of sequential products. This risk cannot be quantitated, and further studies to confirm this finding are desirable.

Discontinue medication pending examination if there is sudden partial or complete loss of vision, or if there is a sudden onset of proptosis, diplopia or migraine. If examination reveals papilledema or retinal vascular lesions medication should be withdrawn.

Since the safety of Ovulen and Demulen in pregnancy has not been demonstrated, it is recommended that for any patient who has missed two consecutive periods pregnancy should be ruled out before continuing the contraceptive regimen. If the patient has not adhered to the prescribed schedule the possibility of pregnancy should be considered at the time of the first missed period.

A small fraction of the hormonal agents in oral contraceptives has been identified in the milk of mothers receiving these drugs. The long-range effect to the nursing infant cannot be determined at this time.

Precautions—The pretreatment and periodic physical examinations should include special reference to the breasts and pelvic organs, including a Papanicolaou smear since estrogens have been known to produce tumors, some of them malignant, in five species of subprimate animals. Endocrine and possibly liver function tests may be affected by treatment with Ovulen or Demulen. Therefore, if such tests are abnormal in a patient taking Ovulen or Demulen, it is recommended that they be repeated after the drug has been withdrawn for two months. Under the influence of progestogen-estrogen preparations preexisting uterine fibromyomas may increase in size. Because these agents may cause some degree of fluid retention, conditions which might be influenced by this factor, such as epilepsy, migraine, asthma, cardiac or renal dysfunction, require careful observation. In breakthrough bleeding, and in all cases of irregular bleeding per vaginam, nonfunctional causes should be borne in mind. In undiagnosed bleeding per vaginam adequate diagnostic measures are indicated. Patients with a history of psychic depression should be carefully observed and the drug discontinued if the depression recurs to a serious degree. Any possible influence of prolonged Ovulen or Demulen therapy on pituitary, ovarian, adrenal, hepatic or uterine function awaits further study. A decrease in glucose tolerance has been observed in a significant percentage of patients on oral contraceptives. The mechanism of this decrease is obscure. For this reason, diabetic patients should be carefully observed while receiving Ovulen or Demulen therapy. The age of the patient constitutes no absolute limiting factor, although treatment with Ovulen or Demulen may mask the onset of the climacteric. The pathologist should be advised of Ovulen or Demulen therapy when relevant specimens are submitted. Susceptible women may experience an increase in blood pressure following administration of contraceptive steroids.

Adverse reactions observed in patients receiving oral contraceptives—A statistically significant association has been demonstrated between use of oral contraceptives and the following serious adverse reactions: thrombophlebitis, pulmonary embolism and cerebral thrombosis.

Although available evidence is suggestive of an association, such a relationship has been neither confirmed nor refuted for the following serious adverse reactions: neuro-ocular lesions, e.g., retinal thrombosis and optic neuritis.

The following adverse reactions are known to occur in patients receiving oral contraceptives: nausea, vomiting, gastrointestinal symptoms (such as abdominal cramps and bloating), breakthrough bleeding, spotting, change in menstrual flow, amenorrhea during and after treatment, edema, chloasma or melasma, breast changes (tenderness, enlargement and secretion), change in weight (increase or decrease), changes in cervical erosion and cervical secretions, suppression of lactation when given immediately post partum, cholestatic jaundice, migraine, rash (allergic), rise in blood pressure in susceptible individuals and mental depression.

Although the following adverse reactions have been reported in users of oral contraceptives, an association has been neither confirmed nor refuted: anovulation post treatment, premenstrual-like syndrome, changes in libido, changes in appetite, cystitis-like syndrome, headache, nervousness, dizziness, fatigue, backache, hirsutism, loss of scalp hair, erythema multiforme, erythema nodosum, hemorrhagic eruption and itching.

The following laboratory results may be altered by the use of oral contraceptives: hepatic function: increased sulfobromophthalein retention and other tests; coagulation tests: increase in prothrombin, Factors VII, VIII, IX and X; thyroid function: increase in PBI and butanol extractable protein bound iodine, and decrease in T^3 uptake values; metyrapone test and pregnanediol determination.

References: 1. Royal College of General Practitioners: Oral Contraception and Thrombo-Embolic Disease, J. Coll. Gen. Pract. *13*:267-279 (May) 1967. **2.** Inman, W. H. W., and Vessey, M. P.: Investigation of Deaths from Pulmonary, Coronary, and Cerebral Thrombosis and Embolism in Women of Child-Bearing Age, Brit. Med. J. 2:193-199 (April 27) 1968. **3.** Vessey, M. P., and Doll, R.: Investigation of Relation Between Use of Oral Contraceptives and Thromboembolic Disease. A Further Report, Brit. Med. J. 2:651-657 (June 14) 1969. **4.** Sartwell, P. E.; Masi, A. T.; Arthes, F. G.; Greene, G. R., and Smith, H. E.: Thromboembolism and Oral Contraceptives: An Epidemiologic Case-Control Study, Amer. J. Epidem. *90*:365-380 (Nov.) 1969.

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Indication—Enovid-E is indicated for oral contraception.

The *Special Note, Contraindications, Warnings, Precautions* and *Adverse Reactions* listed above for Ovulen and Demulen are applicable to Enovid-E and should be observed when prescribing Enovid-E.

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preferred to Basal Cell Nevus Syndrome which might imply relative benignity. Histologically, the nevoid basal cell tumors can not be differentiated from ordinary basal cell epitheliomas. The clinicopathologic differential diagnosis should include nevus cell nevi, seborrheic keratoses, milia, adenoma sebaceum, syringomas and tricho-epitheliomas (epithelioma adenoides cysticum).

Previous authors have suggested the potential use of topically applied 5-FU[4,6] in such cases. Indeed, such treatment appears attractive. However, the twice a day application of 5% fluorouracil solution for 3-6 weeks failed to totally eradicate a single tumor in this case, although occlusion was utilized on selected lesions and significant erythema was observed. It is the authors' opinion that curettage and electrofulguration of early lesions or cryosurgery remain the treatment of choice. If cases are detected early and the tumors are removed when small and noninfiltrating, minimal cosmetic disfigurement results. However, tumor development is relentless and regular follow-up is required. In more advanced cases, Moh's chemosurgery, surgical excision, or plastic repairs may be necessary. X-ray irradiation should be used cautiously as a "last ditch" measure, if at all. A frequent finding in individuals with the Nevoid Basal Cell Epithelioma Syndrome are the so-called DYSKERATOTIC PITS of the palms and soles. These lesions are asymptomatic and present as multiple, 0.1 cm, punched-out, erythematous (or blackened by dirt), non-scaling depressions. These lesions are distinctive and pathognomonic.[7]

As scattered reports of the syndrome appear in the medical literature, other abnormalities become apparent. This is the first case report known to the authors to be associated with cleft lip and palate or the finding of an arm span exceeding the height. The association with a cleft palate may be coincidental but excessive arm span is an unusual finding and complements other reported SKELETAL ABNORMALITIES such as prognathism, bifid or synostotic ribs, scoliosis and kyphosis, spina bifida occulta and brachymetacarpalism.

Other previously described but variably expressed defects involving the ocular, neurologic, and soft tissue were not evident in the case we are reporting. However, some of these abnormalities such as cataracts, glaucoma, agenesis of corpus callosum, medulloblastomas,[8] ectopic calcifications and hyporesponsiveness to parathormone[9] are of significance and more complete tabulations of the Syndrome are contained in the excellent reviews by Tamoney[5] and Gorlin[3] et al.

Genetic studies indicate the mode of inheritance of the Syndrome to be autosomal dominant with marked penetrance and variable expressivity.[10] Most of the studies have indicated a clear-cut family history. However, negative family histories have been reported previously[3] as in our case.

ACKNOWLEDGEMENT

James R. Mabee, D.D.S., Bangor, Maine, referred this case to us.

REFERENCES

1. Gorlin, R. and Goltz, R. W.: *Multiple Nevoid Basal Cell Epithelioma, Jaw Cysts, and Bifid Rib.* New England Journal of Medicine; 262: 908-912, 1960.
2. Archord, H. O.: *Dermatology in General Medicine.* Edited by Fitzpatrick, T. B. et al; McGraw-Hill, 1971, pp 924.
3. Gorlin, R. J. et al: *The Multiple Basal Cell Nevi Syndrome.* Cancer; 18: 89-104, 1965.
4. Tamoney, H. J.: *Basal Cell Nevoid Syndrome.* American Surgeons; 35: 279-283, 1969.
5. Taylor, W. B. et al: *The Nevoid Basal Cell Carcinoma Syndrome.* Arch Derm; 98: 612-614, 1968.
6. Clendenning, W. E.: *Clinical Dermatology.* Edited by Dennis, D. J. et al, Harper & Row, 1972: Vol. 4, 21-20, pp 4.
7. Howell, J. B., Anderson, D. E.; and McClendon: *The Basal Cell Nevus Syndrome.* JAMA; 190: 274-277.
8. Neblett, C. R., Waltz, T. A.; and Anderson, D. E.: *Neurologic Involvement in the Nevoid Basal Cell Carcinoma Syndrome.* J. Neurosurg; 35: 577-584, 1971.
9. Black, J. B. and Clendenning, W. E.: *Parathyroid Hormone Hyporesponsiveness in Patients with Basal Cell Nevi and Bone Defects.*" NEJM; 268: 1157-1161, 1963.
10. Anderson, D. E. and Cook, W. A.: *Jaw Cysts and the Basal Cell Nevus Syndrome.* G. Oral Surgeon; 24: 15-26, 1966.

LENGTH OF HOSPITAL STAYS for common diagnoses and surgical procedures is shortening throughout the U.S., with the western states and particularly California leading the way, according to a recent report of the *California Health Data Corp.* For 14 of 16 specific diagnoses tabulated, stays had shortened over the proceeding 2 years, the only exceptions being diabetes mellitus without acidosis or coma and acute appendicitis with peritonitis, both of which showed a slight increase.

The survey shows that displacement of an intervertebral disk requires a 10.5-day mean stay in California, 10.9 days in other western states, and 12.9 days nationally. Delivery without complications requires a 2.8-day stay in California, 3.1 elsewhere in the west, and 4.0 nationally. Acute myocardial infarction shows 13.9 days in California, 17.7 in other western states, and 22.3 throughout the U.S. — Medical News Report. Vol. 5, No. 43, Oct. 29, 1973.

Hypercyanotic Spells (Cyanotic Crisis) in an Infant With Valvar Pulmonic Stenosis

JOE R. WISE, JR., M.D.*

Some patients with cyanotic congenital heart disease are subject to cyanotic crises characterized by increasing cyanosis, dyspnea and loss of consciousness which may lead to brain damage or death.[1-3] These dramatic spells, variously designated as cyanotic or syncopal attacks, hypercyanotic spells or paroxysmal hyperpnea are a characteristic feature in the history of patients with Fallot's tetralogy.[1-4] The mechanism of these spells is not clear though spasm of the outflow tract of the right ventricle with increase in the right to left shunt is thought to be the cause in some patients.[1-3] Infundibular spasm, however, cannot explain the occurrence of spells in patients with fixed outflow tract obstruction such as valvar pulmonic stenosis or atresia.[3,5] The purpose of this report is to describe the occurrence of hypercyanotic spells in an infant with valvar pulmonic stenosis and to review briefly the mechanism which may account for this.

CASE REPORT

MM, a four-month-old infant boy, was admitted to the hospital for evaluation of cyanosis. His birth weight was 6 pounds eight ounces and his birth, growth and development were normal. Prior to the present illness, he was never cyanotic but the mother "was not happy with his color" and thought it a bit dusky. Intermittent cyanosis was first noted during a diarrheal illness 10 days prior to admission. On the morning of admission shortly after awakening, marked cyanosis developed after 20-30 minutes of crying. During the cyanotic spell which lasted about 20 minutes, he became progressively more short of breath, appeared to be in severe pain and subsequently, listless and limp. He recovered completely after oxygen was administered at a local hospital and was then transferred for further evaluation.

On arrival he was afebrile, weighed 10 pounds 1 and ½ ounces and appeared well grown and fit. The pulse was 140 per minute. The respiratory rate was 50 per minute. Without oxygen, there was moderate cyanosis which was more prominent in the hands and feet than the face. The neck veins could not be seen. The carotid pulse was normal. The precordium was quiet and no thrill was felt. The first heart sound was normal. The second heart sound had only one audible component which was loudest low along the left sternal edge. There was a Grade IV/VI harsh systolic ejection murmur which peaked after mid systole, best heard in the 2nd and 4th interspace. No diastolic murmurs were heard. There was a prominent atrial sound at the left sternal edge. Femoral pulses were normal and not delayed. The electrocardiogram (Fig. 1) showed frontal plane QRS axis of 130 degrees, qR in AVR and in V-1 and prominent P wave suggestive of right atrial enlargement. Chest x-ray showed cardiomegaly with a decreased pulmonary vascular volume. The hemoglobin was 22.3 gms.%, the hematocrit 69.7%, the white count 16.200, and the BUN 5 mg.%.

Shortly after arrival, he developed a typical cyanotic spell which began with irritability and crying and dyspnea and progressed rapidly to intense cyanosis and near coma with agonal gasping respirations. During the spell, the murmur could not be heard. He was placed in the knee-chest position, given morphine and oxygen and recovered and within a few minutes was alert. Cardiac catheterization at Children's Medical Center revealed very severe valvar pulmonic stenosis and a right to left shunt at the level of the foramen ovale (Table 1). Pulmonic valvotomy was performed, the convalescence was uneventful and there has been no further cyanosis. He remains well and active with a residual Grade II/VI systolic ejection murmur and a II/VI early diastolic murmur, both in the pulmonic area.

COMMENT

The typical spell or cyanotic attack in patients with Fallot's tetralogy begins with tachypnea which is progressive and eventually accompanied by deepening cyanosis, limpness and collapse. Convulsions may occur. The systolic murmur becomes short and soft or even inaudible.[3] The attack may last a few minutes or several hours and may vary in frequency from several daily to once in three months. Neither the occurrence nor the frequency of the attacks seems to be related to the degree of cyanosis between attacks, although anemia seems to be a predisposing factor.[1,2] The incidence of these attacks is highest in the first six months of life and they are relatively unusual in children over the age of two.[2] Frequently the attacks occur just after the child has awakened from a long sleep and may be associated with bowel movements, feeding or crying but often are unexpected.[2,4]

Cyanotic attacks are thought to be caused by contraction or spasm of the muscular portion of the right ventricular infundibulum or outflow tract diverting unsaturated blood from the right ventricle through the septal defect into the systemic circulation.[1-3] This contraction of the infundibulum would be teleologically reminiscent of its function in reptiles and amphibians where it acts as a muscular valve contracting in late systole to protect the lungs from the full force of the single ventricle ejecting blood into the aorta.[6] Infundibular spasm alone, however, cannot explain the occurrence of these spells in patients with fixed right ventricular outflow tract obstruction such as pulmonic valvar stenosis or atresia.[5] Guntheroth has suggested that the underlying cause of these spells may be hyperventilation with its attendant increase in venous return to patients who, because of their outflow obstruction, cannot increase the pulmonary blood flow.[4] Ac-

*From Cardiology Section of Medical Service, Eastern Maine Medical Center, Bangor, Maine 04401.

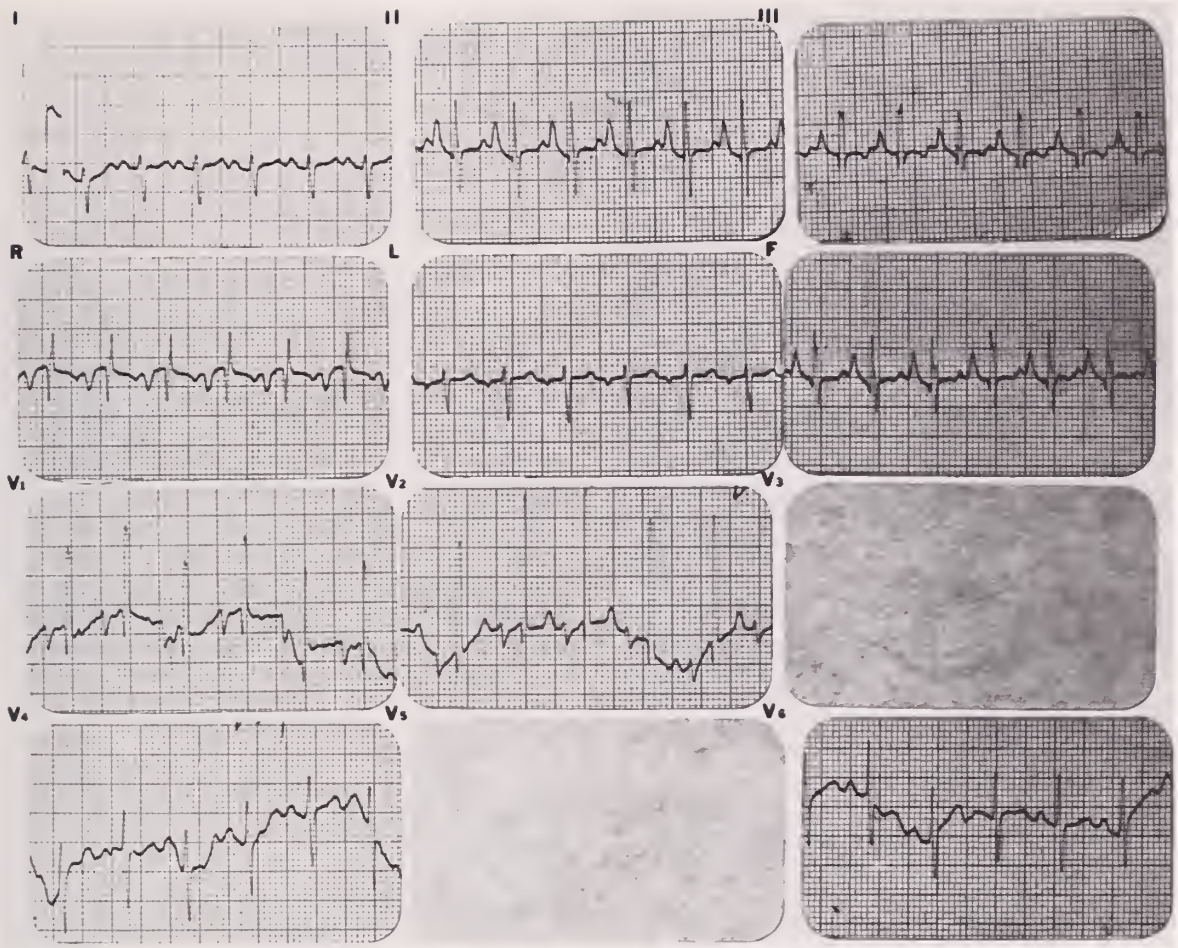

Fig. 1

TABLE 1

SUMMARY OF CARDIAC CATHETERIZATION REPORT

Catheter Position	*Oxygen Saturation Percent*	*Pressure MM Hg*
SVC	35	—
RA	37	A=15 V=8 (mean=7)
RV (Apex)	39	146/11-14
LA	97	A=2 V=7 (mean=4)
LV	57	95/13
FA	54	97/70

Abbreviations: SVC — Superior Vena Cava, RA — Right Atrium, RV — Right Ventricle, LA — Left Atrium, LV — Left Ventricle, FA — Femoral Artery.

cording to this proposal, the infant first begins to hyperventilate, usually upon awakening from a long sleep when the respiratory center seems to be most vulnerable to changes in oxygen and carbon dioxide concentration.[4] The hyperventilation, initiated perhaps by straining or crying or feeding, increases the venous return by the pumping action of the respiratory movements. This increase in cardiac output and presumably venous return, may be as much as 20%.[7] Since these patients with right ventricular outflow tract obstruction cannot increase their pulmonary blood flow, this increased venous return augments the right to left shunt with resultant fall in arterial oxygen saturation. Further hyperventilation follows and a vicious cycle is established. It has been shown that voluntary hyperventilation in patients with tetralogy of Fallot decreases rather than increases the peripheral oxygen saturation as it does in normal subjects.[7]

Alternatively, the increased right to left shunting which occurs with hyperventilation, exercise or excitement may, via decreased oxygen saturation and its direct effect on peripheral arteriales, decrease systemic resistance thereby favoring further right to left shunting and a progressive decrease in arterial saturation.

Although cyanotic spells are most typical of patients with tetralogy of Fallot, they are not limited to this condition and may accompany other forms of cyanotic heart disease with restricted pulmonary blood flow. Spells may occur in patients with

pulmonary and tricuspid atresia and transposition of the great arteries with and without pulmonic stenosis.[8] LaFargue described spells in three patients with pseudotruncus arteriosis — a severe form of tetralogy of Fallot with ventricular septal defect and pulmonary atresia.[5]

In the patients described above, the spells were typical of those occurring in patients with tetralogy of Fallot beginning with uncontrolled crying usually after sleep and characterized by progressive cyanosis, anguish, agonal gasping respirations and collapse. However, signs of right atrial overload such as right atrial gallop (S-4) and EKG evidence of right atrial enlargement are unusual in patients with uncomplicated tetralogy of Fallot perhaps because by venting the right ventricle the ventricular septal defect prevents the marked right ventricular and right atrial pressure overload seen with closed septum pulmonic stenosis. These clinical signs of right atrial overload as well as the differential cyanosis more prominent distally can be of value in the differential diagnosis of patients with cyanosis and cyanotic spells who are otherwise thought to have tetralogy of Fallot. This distinction is an important one since patients with valvar pulmonic stenosis of this severity should have prompt surgical relief, whereas one might be tempted to temporize with a trial of propranolol in a small infant with the tetralogy.[9]

References

1. Braudo, J. L.; Zion, M. M.: (1959) The Cyanotic (Syncopal) Attack in Fallot's Tetralogy. BRIT. M. J. 1: 323.
2. Morgan, B. C.; Guntheroth, W. B.; Blood, R. S.; Fyler, D. C.: (1965) A Clinical Profile of Paroxysmal Hyperpnea in Cyanotic Congenital Heart Disease. Circulation 31: 66-69.
3. Wood, P.: (1958) Attacks of Deeper Cyanosis and Loss of Consciousness (Syncope) in Fallot's Tetralogy. BRIT. Heart J. 20-283.
4. Guntheroth, W. G.; Morgan, B. C.; Mullins, G. L.: (1965) Physiologic Studies of Paroxysmal Hyperpnea in Cyanotic Congenital Heart Disease. Circulation 31: 70-76.
5. LaFargue, R. T.; Vogel, J. H. K.; Pryor, R.; Blount, S. G.: (1967) Psuedotruncus Arteriosis. A Review of 21 Cases with Observations on Oldest Reported Case. Amer. J. Cardio. 19: 239-246.
6. Keith, A.: (1924) Fate of the Bulbis Cordis in the Human Heart. Lancet 11: 1267-1273.
7. Van Lingen, B.; Whidbourne, J.: (1952) Oximetry in Congenital Heart Disease with Special Reference to the Effects of Voluntary Hyperventilation. Circulation 6: 740-748.
8. Fyler, D. C.: Personal Communication.
9. Cumming, G. C.: (1970) Propranolol in Tetralogy of Fallot, Circulation 41: 13-15.

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References

1. Editorial: "Nodule-on-the-leg" Syndrome. New Eng. J. Med. *274*: 463-464, 1966.
2. Jillson, O. F.: Fringe Benefits from Intradermal Skin Testing of the Tuberculin Type. Ann. Allergy *24*: 391-396, 1966.
3. Favour, C. B.: Systemic Manifestations of Erythema Nodosum. California Med. *85*: 207-212, 1956.
4. Lawy, H. S.: Use and Interpretations of the Antistreptolysin Test. Ann. Rheum. Dis. *19*: 42-47, 1960.
5. Medeiros, A. A.; Marty S. D.; Tosh, F. E.; and Chin, T. D. T.: Erythema Nodosum and Erythema Multiforme as Clinical Manifestations of Histoplasmosis in a Community Outbreak. New Eng. J. Med. *274*: 415-420, 1966.
6. Sarner, M. and Wilson, R. J.: Erythema Nodosum and Psittacosis: Report of five cases. Brit. Med. J. *2*: 1469, 1965.
7. Simpson, R. G.: Erythema Nodosum: Provocation Phenomen, with special reference to lymphogranuloma venereum (Nicolas-Favre). Dermatologica *101*: 94-107, 1950.
8. Fry, J.: Familial Erythema Nodosum. Brit. Med. J. *1*: 529-531, 1952.
9. Daniels, W. B., and MacMurray, F. G.: Cat Scratch Disease. Report of 160 Cases. JAMA *154*: 1247, 1954.
10. Editorial: The Kveim Test in Sarcoidosis. Amer. J. Med. *30*: 495-501, 1961.
11. Holcomb, F. C.: Erythema Nodosum Associated with the Use of an Oral Contraceptive. Obstet. Gynec. *25*: 156-157, 1965.
12. Michelson, H. E.: Erythema Nodosum. Arch. Derm. *77*: 546-553, 1958.
13. Lever, W. F.: *Histopathology of the Skin,* 3rd ed., Philadelphia: J. B. Lippincott Company, 1961, pp 116-118.
14. Waisman, M. and Thomas, M. A.: Benign Pulmonary Hilar Lymphadenopathy in Erythema Nodosum. Arch. Derm. *82*: 754-757, 1960.
15. Favour, C. B. and Sosman, M. C.: Erythema Nodosum. Arch. Intern. Med. *80*: 435-453, 1947.
16. Winer, L. H.: Histopathology of Nodose Lesions of Lower Extremities. Arch. Derm & Syph. *63*: 347-357, 1951.
17. Solomon, L. M. and Beerman, H.: Cold Panniculitis. Arch. Derm. *88*: 897-900, 1963.
18. Perry, H. O. and Winkelmann, R. K.: Subacute Nodular Migratory Panniculitis. Arch. Derm. *89*: 170-179, 1964.
19. Gardner, F. H. and Diamond, L. K.: Autoerythrocyte sensitization. A form of purpura producing painful bruising following autosensitization to red blood cells in certain women. Blood *10*: 675-690, 1955.
20. Canizares, O. and Costello, M.: Erythema Nodosum Type of Lepra Reaction. Arch. Derm. *85*: 29-40, 1962.

Cytogenetic Services in Maine

A View to the Future†

KENNETH ALLEN, PH.D.* and JOHN NORTON**

INTRODUCTION

The Cytogenetic Laboratory is the focus of attention of an entire genetic counseling system. From its base, physician education is provided, genetic research is undertaken, new techniques are tested, public information is distributed and genetic counseling is made on the basis of laboratory findings. Although this report talks about the needs for a laboratory service, the larger issue of delivering medical-genetic counseling is implicit. Buccal smears, chromosome karyotypes and amniocentesis are techniques used in the diagnosis of genetic disease. These techniques are being performed in several hospital laboratories in Maine at this time. Like other laboratory techniques, they tend to be unique early in their developmental history. But once there is widespread understanding of any diagnostic tool, it becomes nearly universal in availability. There is every reason to assume that this will be true of the procedures of cytogenetics in the future. First available only in Boston and now at several selected Maine hospitals, soon these procedures will be packaged and offered in the smaller Maine community hospitals.

Once again, the focus of this report is the location of laboratory services that will determine the direction of cytogenetic services in Maine including public and physician education, research, genetic counseling and proficiency testing.[1]

Present Status – Maine

At the present time, there is no program in Maine that provides all of the ingredients for a cytogenetics service indicated above. But there is substantial interest by professionals demonstrating a desire to undertake the provision of these services. Currently, a number of pediatricians and general practitioners use karyotypes as a diagnostic tool. The physicians in Table 1 probably account for the majority of the request for this service.

If one can assume this survey accurately reflects demand for service, karyotyping as a diagnostic tool is used only to a limited extent in Maine.

Amniocentesis is of more limited use in Maine. Of the physicians interviewed, only two had employed this diagnostic technique.

Two areas in Maine indicate a strong commitment to further develop and broaden the current working definition of cytogenetic services in Maine. These include the Maine Medical Center in Portland and the Maine Genetic Counseling Center in Ellsworth.‡ These two centers differ in character and approach; yet each has something important to bring to a cytogenetic service. The Maine Medical Center is the largest, most diversified medical facility in the State. The Maine Medical Center is involved in physician and allied health education, research activities related to patient care, and is in every way a modern medical center. As seen from Table 1, the Maine Medical Center currently accounts for the majority of the karyotypes in Maine per year.

The pediatricians and geneticists involved in the Maine Genetic Counseling Center, Ellsworth, represent an exciting and innovative approach in preventive medicine in an essentially rural setting. Emanating out of a much smaller community setting, Maine Coast Memorial Hospital is willing to provide space for laboratory facilities. Three pediatricians and two geneticists are active in the process of developing the Genetic Counseling Center. The commitment of the personnel involved, plus the close proximity of the Jackson Laboratories with their mammalian genetic interests for a source of consultants will add to the possibilities for future development of cytogenetics in Maine.

Planning Elements

It is estimated that 200 karyotypes per year is a minimum requirement for the maintenance of a viable laboratory.[3] Dr. T. Kushnick[4] estimates that 75% of the cases referred to a laboratory for karyotyping or with observable genetic disorders require genetic counseling. The Wisconsin State Laboratory of Hygiene is a successful example of a highly coordinated program between various state agencies and the cytogenetic service *per se*.[5] The service must be at the focal point coordinating genetic

†The authors of this report would like to express their appreciation for grant support from the Bureau of Health, Department of Health and Welfare; Director, Peter J. Leadley, M.D.

*Professor and Chairman, Department of Zoology, University of Maine, Orono, Consultant to the Project.

**Manager, Research, Evaluation and Planning, Bureau of Health, Department of Health and Welfare, Project Director.

‡Project Director, Sondra Everhart; Medical Director, Morris Lambdin, M.D.; supported by grants from the Bureau of Health, Department of Health and Welfare; Division of Medical Services and The March of Dimes.

TABLE 1[2]

Physicians	*Location*	*Karyotypes/ Year*	*Lab. performing service*
Dr. J. F. Stocks	Maine Medical Center Portland, Maine	60	Same
Dr. H. J. Wheelwright	Augusta General Hosp. Augusta, Maine	5-10	Hartford, Conn.
Dr. Anthony Betts	Thayer Memorial Hosp. Waterville, Maine	15-25	Same, and Medical Maine
Dr. Bernhoff Dahl	Eastern Maine Medical Center Bangor, Maine	3	Boston
Dr. M. A. Lambdin	Maine Coast Memorial Hosp. Ellsworth, Maine	20	Boston
		103-118	

counseling, physician and patient education, as well as genetic evaluation through laboratory investigations and research.

Maine can anticipate 17,000 births per year for the next several years.[6] Abnormal karyotypes will appear at a rate of 66 per 10,000 live births or 115 cases per year in Maine. Three times this number might be an expected case-load for screening and diagnosis or 345 cases per year.[7] All expectant mothers over forty years of age should be encouraged to have amniocentesis and karyotypes to detect chromosomal abnormalities of the fetus (principally, nondisjunction). In 1970, there were 257 pregnancies of women forty or older in Maine.[8] It is often recommended that these procedures be encouraged with women thirty-five and older (in 1970, 1126 babies were born to mothers thirty-five or older[9]). Additionally, it has been determined that 30-40% of all spontaneous abortions in early pregnancies are due to chromosomal abnormalities.[10] Families with a history of spontaneous abortions should be encouraged to have karyotypes made for both parents. Spontaneous abortions can be expected at a rate of *c.* 1.5 per 1,000 births.[11]

From this kind of data, Maine can expect at least 150 karyotypes per year and might reach a peak of several hundred tests if the service were utilized in all the situations cited above. (The numbers are not necessarily additive as mothers over 40, for example, are at risk.) Amniocentesis, on the other hand, is a very specialized technique requiring tissue culture and intensive medical, obstetric, gynecologic and genetic support services. As demand for amniocentesis and amnio acid studies increases, these skills can be developed in Maine. For the present time, however, the volume can be satisfactorily handled in Boston.

A Look to the Future

Genetics and genetic counseling must be able to evaluate and manage patients with a wide range of problems. These problems can require very sophisticated diagnosis and very involved treatment including surgery. The amount of required medical care can vary tremendously; but the socio-economic, psychological or even psychiatric follow-up can last a lifetime. Dr. Joseph Stocks of the Maine Medical Center,[12] when listing the kinds of problems that one is likely to encounter in this program, includes mental retardation, congenital anomalies, anomalies of growth and sexual development, birth defects, chromosomal abnormalities, the FLK or ULK (funny or unusually looking kinder) and finally, the whole spectrum of "inherited disease." This last category includes patient needing evaluation or information about hemophilia, Tay-Sachs disease, cystic fibrosis, diabetes, dwarfism and more general counsel regarding heart attacks, strokes and cancer. These diseases encompass the entire spectrum from child development to general counseling of adults involving marriage and reproduction.

Support of a genetics counseling center is Maine presupposes a long-term financial commitment of individual patients and genetic research into technical areas as well as population genetics determining the "gene burden" of our relatively immobile resident population of nearly one million. It is suggested by other programs across the nation,[13] and within Maine,[14] that State government take a leadership role in coordinating, planning and financing priority items. Clinical medicine, genetics and genetic counseling is only part of a system that includes social work, family counseling, psychology, care of mentally retarded, developmental disabilities, rehabilitation, marriage, family counseling and follow-up are all essential to an effective effort. State government is in a unique position to coordinate these tasks.

General Conclusion

This report deals principally with cytogenetic laboratory services in Maine. The authors recognize that cytogenetic laboratory services presupposes identification of patients at risk, genetic coun-

seling and a whole spectrum of medical and social services to follow. The recommendations that follow deal only with the laboratory service, its location and development.* It is assumed that a well run laboratory service will be the key to a well organized cytogenetic counseling service.

— Maine should have a single cytogenetics program with laboratory services at two locations: the Maine Medical Center, Portland; and the Maine Coast Memorial Hospital, Ellsworth, affiliated with the Maine Genetic Counseling Center.
— Representatives from these two areas should meet soon in Augusta to work out a coordinated program.
— The Maine State Public Health Laboratory should perform periodic quality assessments of the cytogenetic laboratory services. Charles Okey, Ph.D., should seek legislative support to fund this new function.
— Utilization of State resources such as research funding sources and the Jackson Laboratory mammalian genetic interests should be pursued.

These recommendations, if implemented successfully, will insure that immediate physician needs can be met, coordination of cytogenetic counseling services can be accomplished and Maine citizens will receive the benefits of new technology in medicine.

*Recommendations endorsed by the Maine State Comprehensive Health Planning Council, Mark R. Knowles, Executive Director.

NOTE

As the second recommendation requests, Dr. Leadley held a meeting of the principals involved, in Augusta, on Wednesday, October 3, 1973. Minutes of the meeting are available upon request.

REFERENCES

1. Sly, William S., 1973, *What is Genetic Counseling?* Contemporary Genetic Counseling 9 (4) :5-18 National Foundation — March of Dimes, White Plains, N.Y.
2. Personal Survey, Dr. Kenneth Allen.
3. Dr. John Littlefield, Massachusetts General Hospital, Boston, Mass. Personal communication.
4. T. Kushnick, M.D., Department of Pediatrics, College of Medicine, Newark, N.J., personal communication.
5. Stanley Inhorm, M.D., Director, Wisconsin State Laboratory of Hygiene. Personal communication.
6. Maine Vital Statistics. 1960-1970. State of Maine Department of Health and Welfare, Augusta, Maine.
7. Everhart, Sondra, Director, Genetic Diseases Program, the Maine Genetic Counseling, Augusta, Maine, personal communication.
8. *Op. Cit.*, Ref. 6 (1970).
9. *Op. Cit.*, Ref. 6.
10. Stern, Curt, Principles of Human Genetics, 3rd Ed., 1973. W. Freeman and Co., San Francisco, California, pg. 131.
11. Data derived from Kansas Vital Statistics, 1968, comparison with Maine data are estimates.
12. Stocks, Joseph, M.D., Chief Pathologist, the Maine Medical Center, Portland, Maine, personal communication. Many of the ideas encompassed in this section could be attributed to helpful comments from other Maine physicians.
13. See example. Reference 5.
14. Bjorn, John C., M.D., Promis Clinic, Hampden Highlands, Maine. Personal communication.

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ECONOMIC RESTRICTIONS ON PHYSICIANS

Ned F. Parish, President of the National Association of Blue Shield Plans, made the following comments in a report to Blue Shield Plans nationwide. Centering around Phase IV controls, the report demonstrates why a self-enforcing economic control system should be enacted.

Despite the fact that free markets have historically served our society well, non-institutional physicians remain the single professional group still operating under restrictions in the current Phase IV of the Administration's Economic Stabilization Program.

While there remain many emotionally charged arguments for restricting physicians' fees in light of current inflationary trends in medical costs, recent statistics illustrate that the time has now arrived to move toward a more voluntary, self-enforcing system.

Statistics indicate that the nation's physicians have performed conscientiously in controlling prices during the Economic Stabilization Program. During the first three phases of the ESP, physician fees rose less rapidly than the Consumer Price Index "all items" index. During this period, the rate of increase was less than the "medical care" component of the CPI; the annualized first quarter 1973 increase was 1.8 percent.

Clearly, the nation's doctors have made a serious effort to contain those medical costs over which they have had direct control. Accordingly, the present time would perhaps provide an appropriate opportunity to modify these controls.

Though fees might initially show some increase due to the long period of mandatory controls while physicians' costs were still rising, an "equalizing increase" would soon be effected. Reliance upon self-administered physician fee controls would also free the Cost of Living Council to deal with the more pressing problem of hospital costs.

At this point in our sophisticated medical economic milieu, physicians realize that the failure of voluntary controls would guarantee reimposition of mandatory controls.

All evidence indicates that physicians led by responsible organizations such as the American Medical Association, could mount an effective voluntary campaign to restrain profit margins to current levels, emphasize utilization of ambulatory facilities instead of costly inpatient hospital facilities wherever possible, and promote efficiency in the delivery of inpatient hospital services by careful ordering of those tests and procedures which are medically necessary.

Hospital medical staffs would also have a new incentive to review sophisticated purchases of seldom-utilized medical equipment and medical providers would be motivated to work toward the expeditious and responsible implementation of Professional Standards Review Organizations.

The current economic period therefore appears to be a good one to test a more voluntary approach. To remain indefinitely under the current restrictions can only diminish physician support for long-term cost containment programs and other, important administrative initiatives in the healthcare field.

News, Notes and Announcements

Notice!

The Committee on Continuing Education would like to know how many physicians would be interested in attending a course on alcoholism and drug abuse. The proposed course would be given each Wednesday afternoon for a one to two hour period during the months of January, February, or March 1974. The exact place of the course is as yet undetermined, but would probably be in the Portland area. Would all physicians interested in attending such a course please contact either Dr. Richard Chamberlin, Thayer Hospital, Waterville, Maine 04901 or Dr. Alan Elkins, Chief of Psychiatry, Maine Medical Center, Portland, Maine 04102.

International Conference in Ophthalmology and Otolaryngology

Symposia Medica Foundation presents an International Conference on Clinical Problems in Ophthalmology and Otolaryngology to be held in Jerusalem, February 14-24, 1974.

For further information, contact:

Ms. Cynthia Soika, M.A.
Projects Director
Symposia Medica Foundation
305 East 24th Street
New York, N.Y. 10010

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Minutes of the Maine Neurosurgical Society

September 4, 1973

Members present at this meeting were José M. Rodriguez, M.D., Bruce Trembly, M.D., Teodoro C. Dela Cruz, Jr., M.D., Julius E. Ciembroniewicz, M.D., and Daniel A. Rock, M.D. Dr. Ciembroniewicz presented all cases of lumbar discs which have been operated on from April 1972, to March 1973. The charts and x-rays were available and almost all cases were reviewed. A lively discussion followed which included all aspects of treatment of a lumbar disc, but especially the value of multiple disc exploration was discussed in lumbar disc disease which ordinarily involves a single root. During the discussion of chart write-ups, a note explaining the indication for surgery appeared desirable either in the progress notes or in the operative write-up.

The procedure of Barbotage was discussed following a communication by Mr. Merrill of Maine Blue Cross and Blue Shield. There was general agreement that this procedure of Barbotage is not an accepted procedure, and any fee allowance might be similar to that for a lumbar puncture.

Also discussed were customary and reasonable charges for the unilateral and bilateral angiography. Although bilateral carotid angiography follows unit values recommended by California Relative Value Studies, many members have used independent values for brachial angiography when combined with a left carotid angiography.

With relation to epileptics and driving licenses, the members present recommended that the practice of examination by a specialist in neurology should be continued and electroencephalogram would appear to be a reasonable requirement especially in those parts of the State where electroencephalography can be readily obtained. The determination of a seizure-free period adequate to permit the safe driving of a motor vehicle should be left on an individual basis. At no time should a physician be required to report the presence of epilepsy, since such procedures cause epileptics to withhold the disclosure of their disability and in turn prevent proper treatment.

The next meeting of the Society will be held in Lewiston in November.

Daniel A. Rock, M.D.
President

Maine Specialist Chosen as Fellow in Cardiology Organization

A Maine doctor has been designated as a Fellow of the American College of Cardiology (ACC), the national organization representing specialists in cardiovascular diseases. He is among a group of 60 specialists admitted in the first half of 1973 to the medical specialty society's highest membership category.

Harold L. Osher, M.D., Portland, Maine, ACC Governor for the State of Maine, indicated that the new Fellow is Robert P. Kraunz, M.D. of Lewiston.

Dr. Kraunz, as well as the other new ACC Fellows, has fulfilled stringent membership requirements based on years of practice and specialty certification. This effort, according to Dr. Osher, culminates in his being considered by colleagues in his community as a specialist or consultant in cardiovascular diseases.

County Society Notes

KENNEBEC

The Kennebec County Medical Association met at the Silent Woman Restaurant in Waterville, Maine on September 20, 1973. A pleasant social hour and buffet dinner were followed by the business meeting, conducted by the President, Dr. Richard R. Dole.

A letter from the Maine Medical Association was read announcing the date of the fall meeting (Dec. 9) and asking for nominees for Standing and Special Committees.

Dr. Richard T. Chamberlin, Executive Committee member, requested greater input from the county association members to their delegates on issues which will be considered at the State Medical Association level.

There was no old business.

Under new business, Dr. Dole introduced Mr. John LaCasse, Director of the Kennebec Valley Regional Health Agency, who described the Early Periodic Screening Diagnosis and Treatment program which his agency is conducting in Kennebec and Somerset counties. Mrs. Jeanette Leighton, R.N., nursing director for the agency, amplified the presentation in the question and answer period.

The main speaker of the evening, Dr. Lawrence Crane, Director of the Orthopedic Service of the Maine Medical Center, gave an interesting talk on "Total Joint Replacement." On the basis of his experience, he indicated that approximately 95 percent of hip joint replacements are successful. Although knee and shoulder joint replacement are less successful, the use of metal and plastic in the surgical reconstruction of these areas represents a distinct advance in the therapy of incapacitating joint disease.

The meeting was adjourned at 8:50 p.m.

KEVIN HILL, M.D., *Secretary*

Letters to the Editor

To the Editor:

At age sixty-five, many chairmen and other members of the faculty of medical schools are required to relinquish their positions and develop a new modus vivendi. Some continue to be productive along academic lines but others may find opportunities lacking and, as a result, "wither on the vine." It was because of this tremendous waste of manpower that Dr. Joseph Moldaver of New York City was stimulated into initiating the Senior Medical Consultants several years ago. The list of those consultants read like the "Who's Who" of American Medicine. With funds obtained from the Federal government and from the hospitals which have benefited from the efforts of these senior medical teachers, honoraria have been established. Over 70 hospitals in New York, New Jersey and Connecticut have had their teaching programs enhanced by some 35 consultants. In many instances, the experience gleaned by the staffs of these hospitals has been so rewarding that they have requested weekly visits by the same consultants on a continuing basis. I believe that there are many retired physicians in Maine who have had extensive careers in teaching, writing, research and serving as administrators and who would be most eager to participate in programs of this type. One of the requirements for retiring gracefully is to continue to be productive, and there is no more gratifying experience than to be able to contribute to the knowledge of your colleagues. Those retired physicians who have enjoyed academic backgrounds and who are eager to participate in this program are urged to get in touch with Dr. Alfred Hurwitz, Liberty, Maine 04949.

It is hoped that we can do for our State of Maine as much as Dr. Moldaver has been able to accomplish in New York, New Jersey and Connecticut. The need here is certainly great.

ALFRED HURWITZ, M.D.
Liberty, Maine 04949

To the Editor:

NEGLECTED NOOKS AND CRANIES

It is quite logical that a colloquialism may be misleading as a subject for a professional paper; however, continued reading will reveal the logic and its simplicity I'm sure.

I cannot recall in my years of doing Obs. and Gyn, such extreme evidence of symptoms and justifiable complaints of cystitis, urethritis, labiitis, vaginitis, and of course, cervicitis. The influx of teenagers with their multiplicity of complaints, their descriptions in a very naive conversation prompts one to carefully and diplomatically inquire as to how much, if any, vaginal hygiene instruction has been taught them at home. One is taught how and when to clean one's teeth early in childhood. Babies are housebroken, but does the Mother ever go far enough to teach the child proper laundering at stool?

And so it is with these little teenage girls afflicted with E. Coli Trichomonas, Monoliasis, etc., which has set up an invasion over the months and about which nothing is done until taken to the Doctor. I find that the advice and therapy is quite entirely inadequate to appease the burning, itching irritability of this affliction.

I recall very vividly in my practice what a tremendous encumbrance a physician encountered in prescribing periodic douches. By a large percentage of my practice it was frowned upon, feeling that it had a contraceptive flavor.

The many cases I have treated in the last two years, quite a few had been treated elsewhere rather unsuccessfully. The tablets so commonly used are a very poor substitute for Sultrin® cream which is quite a specific when used 7-10 days preceded by a cleansing douche of plain warm water, if the patient isn't sulfa sensitive.

I believe the big mistake made by the majority of therapists is to entirely disregard the partner or the husband as the case may be. He can be successfully treated with Flagyl®. Otherwise the treatment amounts to nothing as the infection is passed back and forth, much to the discouragement of the patient as well as the Doctor.

Recently there were two cases in the office with a most unattractive Trichomonas infection, both of whom exhibited a strong suggestion of low grade P.I.D., which responded beautifully to Sultrin and appropriate dosage of Penicillin. On their return for appraisal of therapy, all lower quadrant tenderness had disappeared. I believe if courteously and diplomatically handled, these deserving patients can be very decidedly minimized by the Family Doctor.

My desire in this paper is to try to interest and influence the GP and the FD who are nearer to these cases, to project a little more influence into their families in revealing to them how simple hygiene of this type can become and without embarrassing the Mother or daughter. And, of course, this type of treatment practically neutralizes the chances of primary sterility when they have reached the age of matrimony, and become so frustrated in their desire to start their families.

REGINALD T. LOMBARD, M.D.
793 Main Street
South Portland, Maine 04106

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ANES	Anesthesiology
AM	Aerospace Medicine (special field of Preventive Medicine)
CD	Cardiovascular Disease (sub-specialty of Internal Medicine)
CHP	Child Psychiatry (sub-specialty of Psychiatry)
CRS	Colon and Rectal Surgery
D	Dermatology
DR	Diagnostic Roentgenology (special field of Radiology)
FOP	Forensic Pathology (special field of Pathology)
FP	Family Practice
GE	Gastroenterology (sub-specialty of Internal Medicine)
GPM	General Preventive Medicine (special field of Preventive Medicine)
GS	General Surgery
IM	Internal Medicine
NS	Neurological Surgery
N	Neurology
OBG	Obstetrics and Gynecology
OM	Occupational Medicine (special field of Preventive Medicine)
OPH	Ophthalmology
ORS	Orthopedic Surgery
OTO	Otolaryngology
PATH	Pathology
PD	Pediatrics
PDA	Pediatric Allergy (sub-specialty of Pediatrics)
PDC	Pediatric Cardiology (sub-specialty of Pediatrics)
PMR	Physical Medicine and Rehabilitation
PS	Plastic Surgery
P	Psychiatry
PH	Public Health (special field of Preventive Medicine)
PUD	Pulmonary Diseases (sub-specialty of Internal Medicine)
R	Radiology
TR	Therapeutic Radiology (special field of Radiology)
TS	Thoracic Surgery
U	Urology
OO	Unspecified (retired, not in practice, no specialty reported)
99	Other

Members

Active–Honorary–Senior–Affiliate–Junior–Service
(Corrected To January 1, 1973)

ANDROSCOGGIN COUNTY

President – Gilbert R. Grimes, M.D.

Secretary-Treasurer – Richard M. Swengel, M.D.

ACTIVE

Adams, Patricia Ann (PD) Freeport, 04032
Amfilo, Basil (ANES) 626 Main St., Lewiston 04240
Anderson, Donald L. (U,GS) 369 Main St., Lewiston 04240
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Beliveau, Bertrand A. (IM) 56 Howe St., Lewiston 04240
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Goodwin, Ralph A., Jr. (OPH) 33 Court St., Auburn 04210
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Mendes, Joseph M. (FP,IM) 5 School St., Lisbon Falls 04252
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Milazzo, John (FP) 42 Elm St., Auburn 04210
Miller, Clark F. (R) Greene 04236
Morin, Gerard L. (GS) 185 Webster St., Lewiston 04240
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Nadeau, J. Paul (FP) 91 Pine St., Lewiston 04240
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Young E. Stanley (FP) Poland Spring 04274
Zanca, Ralph (IM,99) 185 Webster St., Lewiston 04240

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Williams, James A. (FP) 39 Pleasant St., Mechanic Falls 04256

SENIOR

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Rand, Carleton H. (ORS) 219 Oak St., Lewiston 04240
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AROOSTOOK COUNTY

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Secretary – Benoit Ouellette, M.D.

Treasurer – Clyde I. Swett, M.D.

ACTIVE

Albert, Rodrigue J. (PD) 9 Pleasant St., Fort Kent 04743
Albro, Ward A. (ANES) County Rd., Houlton 04730
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Chien, Chang-chi (ANES) 15 Teague St., Caribou 04736
Chow, Alroy A. (IM) 162 Academy St., Presque Isle 04769
Collins, H. Douglas (IM) Caribou Clinic, Caribou 04736
Curtin, Daniel C. (IM) 555 Main St., Presque Isle 04769
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Dunham, Marguerite C. (PH) R.F.D. No. 1, Dresden 04342
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Gregory, Frederick J. (GS) 504 Main St., Caribou 04736
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Hamlin, Paul S. (U) 122 Academy St., Presque Isle 04769
Harrison, George J. (FP) Market Square, Houlton 04730
Hayward, I. Mead (PD) So. Main St., Caribou 04736
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Labbe, Onil B. (FP) Van Buren 04785
Madigan, John B. (FP,ANES) Houlton 04730
Mazerolle, Denis R. (OBG) 228 Sweden St., Caribou 04736
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Nicholas, Eric F. (FP) Mars Hill 04758
O'Brien, William A. (R) Arthur R. Gould Mem Hosp., Presque Isle 04769
Ocana, Emilio (FP) No. Main St., Ashland 04732
Ouellette, Benoit (FP,OBG) 1 James St., Fort Kent 04743
Page, Rosario A. (GS) RFD No. 2, Caribou 04736
Pendleton, Arthur D. (FP,ANES) 3 Green St., Fort Fairfield 04742
Pines, Philip (FP,GS) Maine St., Limestone 04750
Price, Richard D. (ANES) R.F.D. 2, Caribou 04736
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Smith, Marshall E. (P) Pratt Rd., Caribou 04736
Somerville, Robert B. (GS) 45 Hillside St., Presque Isle 04769
Tao, Zui S. (IM) Main St., Fort Kent 04743
Wakana, Minoru (PATH) 33 Lyndon St., Caribou 04736
White, Leland M. (IM,A) 18 Pleasant St., Caribou 04736
Williams, Edward P. (P.A.)-(FP,OBG) 72 Main St., Houlton 04730
Wilson, G. Ivan (IM) 48 Court St., Houlton 04730
Yaghmai, Madjid (R) Cary Mem. Hosp., Caribou 04736

HONORARY

Boone, Storer W. (FP,GS) 54 Third Ave., Presque Isle 04769
Kirk, William V. (FP,GS) Eagle Lake 04739

SENIOR

Brown, Stephen S. (FP) Mars Hill 04758
Swett, Clyde I. (GS) 18 Sherman St., Island Falls 04747

AFFILIATE

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Toussaint, Leonid G. (FP,CD) Box 9, Fort Kent 04743

JUNIOR

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CUMBERLAND COUNTY

President – Douglas R. Hill, M.D.

Secretary-Treasurer – Alfred E. Swett, M.D.

ACTIVE

Abourjaily, Georges S. (GS) 111 Westcott Rd., South Portland 04106
Adams, David L. (C) 131 Chadwick St., Portland 04102
Adams, Marvin C. (OTO) 52 Gilman St., Portland 04102
Agan, Robert W. (ANES) 144 State St., Portland 04101
Akerberg, Ake (P,N) Lanewood, Cumberland Foreside 04110
Allen, Donald E. (PS) 25 Bramhall St., Portland 04102
Allport, Robert B. (PD) Box C, Pownal 04069
Anderson, John B. (FP) Dudley Coe Inf., Bowdoin College, Brunswick 04011
Andrews, Anneliese M. (ANES) Maine Medical Ctr., Portland 04102
Ansell, Harvey B. (D) 39 Deering St., Portland 04101
Applin, Hilton H. (IM,99) 6 Cumberland St., Brunswick 04011
Aranson, Albert (IM,PUD) Maine Medical Ctr., Portland 04102
Asali, Louis A. (GS) 29 Deering St., Portland 04101
Augur, Newell A., Jr. (IM) 175 Vaughan St., Portland 04102
Austin, William H. (IM,99) 125 Chadwick St., Portland 04102
Bacastow, Merle S. (IM) York Hosp., York, Pa. 17405
Baldini, Elio (ANES) 22 Bramhall St., Portland 04102
Baldwin, Warren C. (OBG) 42 Deering St., Portland 04101
Barnes, Kirk K. (GS) Baribeau Dr., Brunswick 04011
Barron, Martin A., Jr. (PD) 229 Vaughan St., Portland 04102
Bennert, Harry W., Jr. (OBG) 7 Bramhall St., Portland 04102
Bennet, Eben T. (OBG) 49 Deering St., Portland 04101
Berkovitch, Sumner (PD) 229 Vaughan St., Portland 04102
Bettle, Ronald A. (GS) Parkview Professional Bldg., Brunswick 04011
Bidwell, Robinson L. (NS,N) 31 Bramhall St., Portland 04102
Binette, Germain A. (R) Webber Hosp., Biddeford 04005
Bliss, Harry A. (CD) 39 Deering St., Portland 04102
Bonney, James H. (IM) 53 Chadwick St., Portland 04102
Bove, Louis G. (IM) 233 Vaughan St., Portland 04102
Bowman, Peter W. (CHP,P) 56 Baribeau Dr., Brunswick 04011
Boyd, Marjorie A. (HEM) 13 Charles St., Portland 04102
Branson, Sidney R. (FP) 37 Main St., So. Windham 04082
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Briggs, Winton (IM) 291-A Ocean House Rd., Cape Elizabeth 04107
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Britton, Richard C. (GS,VS) Maine Medical Ctr. Portland 04102
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Brown, Douglas H. (FP) 1 Birchwood Rd., Cape Elizabeth 04107
Bryant, Daniel C. (IM) 233 Vaughan St., Portland 04102
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Burke, John N. (OBG) 144 State St., Portland 04101
Burnett, Claude A., Jr. (GS,TS) 59 Deering St., Portland 04101
Burnham, Harold N. (FP) 130 Main St., Gorham 04038
Burns, Robert M. (FP) Box 151, Westbrook 04092
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Capron, Charles W. (R) 22 Bramhall St., Portland 04102
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Shindo, Hizuchi-Machi, Yawatahama City, Ehime Prefecture, Japan
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Loring, William E. (PATH) 7 Riverside Dr., Falmouth Foreside 04105
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Mack, Francis X. (ANES) 144 State St., Portland 04101
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MacVane, William L., Jr. (GS,TS) 211 State St., Portland 04101
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Marshall, Donald F. (U) 25 Bramhall St., Portland 04102
Martin, Ralf (IM,CD) 131 Chadwick St., Portland 04102
Martin, Thomas A. (ORS) 157 Pine St., Portland 04102
Matthews, Edward C. (PD,PDC) 229 Vaughan St., Portland 04102
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Sigafoos, J. Harvey (ANES)	Pleasant Point	04563
Sube, Janis (GS)	108 Elm St., Camden	04843
Ward, William W. (GS,99)	Box 646, Rockland	04841
Warren, Henry S. (FP)	Derby Rd., Islesboro	04848
Wasgatt, Wesley N. (FP)	41 Talbot Ave., Rockland	04841
Waterman, Dorothy (FP)	Waldoboro	04572
Waterman, Richard (FP)	Main St., Waldoboro	04572
White, Henry O. (GS)	22 White St., Rockland	04841
Williams, Thomas W. (IM)	22 White St., Rockland	04841
Woodruff, Alan F. (IM)	16 Summer St., Rockland	04841
Worthing, Verla E. (ANES)	Box A, Thomaston	04861

HONORARY

Campbell, Fred G. (FP)	Box 484, Warren	04864
Loewenstein, George (OO)	1007 Woodside Dr., Clearwater, Fla.	33516
Naumer, Harry A. (OO)	Lands' End, Port Clyde	04855
Saunders, Sallie H. (OO)	R.F.D., Camden	04843
Stimson, Barbara B. (ORS)	Star Rt. 22-282 Owl's Head	04854

SENIOR

Apollonio, Howard L. (OO)	Box 34, Rockport	04856
Bean, Achsa M. (OO)	Star Route 32, Owl's Head	04854

AFFILIATE

Dennison, Frederick C. (IM)	3 Gillchrest St., Thomaston	04861
Jones, Paul A., Sr. (N,P)	General Delivery, Union	04862
Tounge, Harry G., Jr. (FP)	12 Union St., Camden	04843

LINCOLN-SAGADAHOC COUNTY

President – Peter A. Evans, M.D.

Secretary-Treasurer – George W. Bostwick, M.D.

ACTIVE

Akar, Hamdi (IM,CD)	37 Oak St., Bath	04530
Andrews, John F. (FP)	67 Oak St., Boothbay Harbor	04538
Avantaggio, Frank O., Jr. (GS)	Bristol Rd., Damariscotta	04543
Bachrach, Louis (IM)	17 Meadowbrook Rd., Brunswick	04011
Belknap, Samuel L. (FP)	Damariscotta	04543
Blackburn, Nelson P. (PATH)	Bath Memorial Hosp., Bath	04530
Bostwick, George W. (FP)	Box 388, Newcastle	04553
Burden, Charles E. (PD)	1 North St., Bath	04530
Dixon, Robert H. (OTO)	6 Oak Grove Ave., Bath	04530
Doble, Miriam (FP,ANES)	990 Washington St., Bath	04530
Dorogi, Louis V. (GS)	Old Post Rd., Rt. 138, Bowdoinham	04008
Dougherty, John F. (FP)	112 Front St., Bath	04530
Evans, Peter A. (IM)	65 Baribeau Dr., Brunswick	04011
Fichtner, Paul A. (FP)	10 Oak Grove Ave., Bath	04530
Galen, Robert S. (R)	6 Breckan Rd., Brunswick	04011
Gregory, Philip O. (FP,GS)	St. Andrews Hosp., Boothbay Harbor	04538
Griffin, Carl R., Jr. (GS)	61 Atlantic Ave., Boothbay Harbor	04538
Hill, David S. (FP)	10 Oak Grove Ave., Bath	04530
Horstman, Anthony J. (FP)	McKown St., Boothbay Harbor	04538
Hudson, Henry A. (R)	Southport Island	04569
Keating, Anthony J. (FP)	10 Oak Grove Ave., Bath	04530
Kinder, Edward L., Jr. (GS)	1027 Washington St., Bath	04530
Leck, Richard C. (PATH)	Bath Mem. Hosp., Bath	04530
McGuire, Peter F. (FP)	Peary Dr., Brunswick	04011
Morrison, Charles C. (FP)	Damariscotta	04543
Oceretko, Arkadij (GS)	Gov. King Oak Grove Ave., Bath	04530
Powell, Ralph C. (FP)	Damariscotta	04543
Proctor, Thomas E. (FP,GS)	Boothbay Harbor	04538
Rowan, Gilbert R. (FP)	10 Oak Grove Ave., Bath	04530
Schall, David W. (FP)	56 Baribeau Dr., Brunswick	04011
Sieling, Walter H., Jr. (IM)	Upper Round Pond Rd., Bristol	04539
Smith, Jacob (FP,ANES)	709 High St., Bath	04530
Smith, James O. (FP)	118 Front St., Bath	04530
Starks, Pauline G. (ANES)	Pemaquid Point	04561
Stetkevych, Alexander G. (P)	1 Meadow Rd., Chatham, N. J.	07928
Stewart, Richard D. (PD)	1 North St., Bath	04530
Swanson, Ronald A. (R)	Regional Mem. Hosp., Brunswick	04011
Tracy, Mary J. (PD)	Bristol Rd., Damariscotta	04543
Winchenbach, Francis A. (GS)	910 Washington St., Bath	04530
York, Elihu (IM)	65 Baribeau Dr., Brunswick	04011

HONORARY

Dalrymple, Sidney C. (OO)	So. Great Rd., So. Lincoln, Mass.	01751

SENIOR

Bachulus, John M. (OO)	3 Breckan Rd., Brunswick	04011
Hamilton, Virginia C. (OO)	South Harpswell	04079
Sherman, Fuller G. (OO)	Spruce Pt., Boothbay Harbor	04538

AFFILIATE

Fite, Marcia (OO)	Pemaquid Point	04558

JUNIOR

Evans, Richard, III (IM)	Maine Medical Ctr., Portland	04102

SERVICE

Zeller, Alan W. (GS)	Main St., Newcastle	04553

OXFORD COUNTY

President – Hagop Halladjian, M.D.

Secretary-Treasurer – Stephen B. Dewing, M.D.

ACTIVE

Aucoin, Peter B. (FP)	151 Franklin St., Rumford	04276
Bean, H. Richard (FP,ANES)	121 Main St., Norway	04268
Dewing, Stephen B. (R)	R.F.D. No. 2 Harrison	04040
Dixon, Walter G. (ORS,99)	16 Deering St., Norway	04268
DuMais, Alcid F. (GS)	73 High St., South Paris	04281
Edmond, James A. (GS)	Star Route, Rumford Center	04276
Egan, John F. (ORS)	810 Penobscot St., Rumford	04276
Elsemore, Dexter E. (FP,GS)	11 Main St., Dixfield	04224
Fenger, John R. (GS,99)	241 Main St., Norway	04268
Frigault, Emile J. (FP,R)	Main St., Dixfield	04224
Ganguli, Adwaita K. (U)	191 Lincoln Ave., Rumford	04276
Gorayeb, Eugene (FP,GS)	82 Maine Ave., Rumford	04276
Halladjian, Hagop (PATH)	Rumford Com. Hosp., Rumford	04276
Hamilton, Kenneth G. (GS)	163 Main St., Norway	04268

Handanos, Vassilios (PD)	191 Lincoln Ave., Rumford	04276
Harper, Harry L. (CD)	17 Main St., So. Paris	04281
Hazelton, Warren C. (FP)	2 E. Main St., So. Paris	04281
Hiebert, Joelle C., Jr. (GS)	Box 148, Norway	04268
Jackson, Norman M. (CD)	9 Franklin St., Rumford	04276
Li, Tsung H. (FP)	High St., Buckfield	04220
Makin, John B., Jr. (OBG)	82 Maine Ave., Rumford	04276
Martin, Joseph E. (FP)	35 Main St., Mexico	04257
Moore, Beryl M. (FP)	High St., Oxford	04270
Nangle, Thomas P. (FP)	West Paris	04289
Oestrich, Alfred (FP)	25 Hartford St., Rumford	04276
Phillips, David L. (GS)	191 Lincoln Ave., Rumford	04276
Rowe, Linwood M. (R)	Rumford Com. Hosp., Rumford	04276
Royal, Albert P., Jr. (FP,GS)	82 Maine Ave., Rumford	04276
Rynne, Michael V. (FP)	191 Lincoln Ave., Rumford	04276
Schnittke, Sidney M. (FP,A)	Porter Ave., W., Rumford	04276
Tai, Tse-Wu (ANES)	Rumford Com. Hosp., Rumford	04276
Young, John (FP)	Paradise Rd., Bethel	04217

HONORARY

Howard, Henry M. (FP)	105 Franklin St., Rumford	04276
MacDougall, James A. (FP,D)	303 Penobscot St., Rumford	04276
Nelson, Chesley W. (FP)	8 Nevers Ave., Norway	04268

PENOBSCOT COUNTY

President – Benjamin L. Shapero, M.D.

Secretary – Robert L. Burdick, M.D.

Treasurer – Philip R. Kimball, M.D.

ACTIVE

Adams, Winford C. (FP)	14 Starlight Dr., Brewer	04412
Andrews, Robert P. (R)	489 State St., Bangor	04401
Archambault, John L. (FP)	Christian Hill, R.F.D. No. 1, Winterport	04496
Babcock, Edward B. (IM)	431 State St., Bangor	04401
Ballesteros, Ernesto G. (NS)	292 Hammond St., Bangor	04401
Barrett, Robert J., Jr. (A,D)	Cor. Union & James Sts., Bangor	04401
Bjorn, John C. (FP)	Hampden Highlands	04445
Blackwell, William M. (R)	Millinocket Com. Hosp., Millinocket	04462
Blaisdell, William B. (OPH,OTO)	209 State St., Bangor	04401
Boone, Alan W. (IM)	111 State St., Bangor	04401
Bouton, Dale C. (ORS)	157B Broadway, Bangor	04401
Bragg, Franklin E., II (IM)	336 Mt. Hope Ave., Bangor	04401
Brito, Joseph S. (R)	255 Hammond St., Bangor	04401
Brown, Eugene E. (A,D)	57 Summit Ave., Bangor	04401
Brown, Lloyd (GS,TS)	186 State St., Bangor	04401
Brown, Robert H. (ORS)	MRC, Box 45, Bangor	04401
Burdick, Robert L. (R)	St. Joseph Hosp., Bangor	04401
Burger, Charles S. (FP)	Hampden Highlands	04445
Burke, Paul W. (FP)	5 High St., Newport	04953
Butterfield, Wilfred I. (FP)	119 Main St., Lincoln	04457
Chase, George O. (PATH)	297 Center St., Bangor	04401
Chason, Sidney (OBG)	128 Broadway, Bangor	04401
Clement, James D., Jr. (GS)	77 Essex St., Bangor	04401
Clough, Dexter J., 2nd (OPH)	224 State St., Bangor	04401
Clough, Herbert T. (AM)	R.F.D. No. 1, Box 132, Orrington	04474
Coulton, Donald (OBG,99)	326 State St., Bangor	04401
Cross, Harold D. (FP)	Main Rd. & Summer St., Hampden Highlands	04445
Curran, Edward L. (GS)	209 State St., Bangor	04401
Cutler, Lawrence M. (IM)	31 Grove St., Bangor	04401
Dahl, Bernhoff A. (PATH)	Eastern Maine Med. Ctr. Bangor	04401
Desjardins, Richard F. (FP)	200 Spruce St., Millinocket	04462
Dietrich, Mary M. (IM,PD)	Box 93, Orrington	04474
Dixon, Charles E. (GS)	431 State St., Bangor	04401
Duffey, Richard V. (ORS)	57 Washington St., Bangor	04401
Emery, Frederick C. (PD)	242 Cedar St., Bangor	04401
Emmett, Peter A. (EMER.MED.)	489 State St., Bangor	04401
Emmett, Storer W. (FP)	Eastern Maine Med. Ctr., Bangor	04401
Evans, Stanley J. (IM)	336 Mt. Hope Ave., Bangor	04401
Eyerer, Rudolf E. (PATH)	489 State St., Bangor	04401
Feeley, J. Robert (U)	438 Garland St., Bangor	04401
Fergus, Andrew (P,N)	336 Mt. Hope Ave., Bangor	04401
Field, Richard L. (FP)	489 State St., Bangor	04401
Files, George E. (U)	454 State St., Bangor	04401
Gaillard, Richard A. (OTO)	276 State St., Bangor	04401
Gilman, Herbert C. (FP)	200 Spruce St., Millinocket	04462
Graves, Robert A. (FP)	Sunset Drive, Orono	04473
Hall, Walter L. H. (FP,GS)	130 Middle St., Old Town	04468
Hamlin, Irvin E. (FP)	Main St., E. Millinocket	04430
Hill, Allison K. (GS)	431 State St., Bangor	04401
Holzwarth, Hans A. (IM)	336 Mt. Hope Ave., Bangor	04401
Houlihan, John S. (IM)	209 State St., Bangor	04401
Hudson, Mary H. H. (IM)	110 Spring St., Dexter	04930
Hughes, Edward J., Jr. (PD)	336 Mt. Hope Ave., Bangor	04401
Hunter, Philip G. (GE)	35 Second St., Bangor	04401
Irwin, Carl W. (NS)	336 Mt. Hope Ave., Bangor	04401
Jillson, Otis F. (D,A)	478 State St., Bangor	04401
Kawamura, Takeo (P.A.)-(P)	336 Mt. Hope Ave., Bangor	04401
Kellogg, Robert O. (IM)	431 State St., Bangor	04401
Kimball, Philip R. (ORS)	336 Mt. Hope Ave., Bangor	04401
Kittredge, Francis I. (N)	109 State St., Bangor	04401
Lathi, Dewayani C. (PATH)	220 Randolph Dr., Bangor	04401
Lathi, Subhashchandra G. (R)	255 Hammond St., Bangor	04401
Leonidas, Leonardo L. (PD)	12 Acme St., Brewer	04412
Madaras, John (FP,P)	Bangor State Hosp., Bangor	04401
Manter, Wilbur B. (CD)	1 Fern St., Bangor	04401
Mason, Peter H. (GS)	Millinocket Com. Hosp., Millinocket	04462
Maunz, Don L. (GS)	186 State St., Bangor	04401
McEvoy, Charles D., Jr. (P.A.)-(GS,TS)	186 State St., Bangor	04401
McGinn, John F. (ORS)	205 French St., Bangor	04401
McLean, Preston A. (OBG)	336 Mt. Hope Ave., Bangor	04401
McQuoid, Robert M. (OTO,OPH)	39 Columbia St., Bangor	04401
Meltzer, Jack N. (IM,CD)	128 Broadway, Bangor	04401
Memmelaar, Joseph E. (U)	431 State St., Bangor	04401
Merriam, Thornton W., Jr. (IM)	431 State St., Bangor	04401
Metz, Gerald A. (OPH)	336 Mt. Hope Ave., Bangor	04401
Miragliuolo, Leonard G. (GS)	10 Maple St., Bangor	04401
Moulton, Gardner N. (OPH)	5 Grove St., Bangor	04401
Munce, Richard T. (GS)	336 Mt. Hope Ave., Bangor	04401
Nesin, Bourcard (FP)	21 Penobscot Ave., Howland	04448
Netland, Anders T. (OBG)	431 State St., Bangor	04401
O'Kane, Francis R. (FP,ANES)	200 Spruce St., Millinocket	04462
Ordway, John A. (P)	R.F.D. No. 3, Box 199B, Bangor	04401
Osler, Jay K. (OPH)	74 Birch St., Bangor	04401
Palmer, Thomas H., Jr. (GS)	431 State St., Bangor	04401
Parrot, Hadley (IM)	431 State St., Bangor	04401
Pasternak, Irwin M. (P)	230 French St., Bangor	04401
Patten, Roy S. (IM)	336 Mt. Hope Ave., Bangor	04401
Pearson, John J. (FP)	100 S. Main St., Old Town	04468
Phillips, Lewis E. (IM)	336 Mt. Hope Ave., Bangor	04401
Porter, Edward C. (R)	489 State St., Bangor	04401
Purinton, William A. (OBG)	St. Joseph Hosp., Bangor	04401
Schroder, John C. (P.A.)-(OTO)	205 French St., Bangor	04401
Sensenig, David M. (GS,TS)	431 State St., Bangor	04401
Sewall, Elmer M. (FP)	14 Park St., Orono	04473
Shapero, Benjamin L. (PD)	431 State St., Bangor	04401
Shubert, Alice J. (OBG)	125 Leighton St., Bangor	04401
Shubert, William M. (OBG)	336 Mt. Hope Ave., Bangor	04401
Shurman, Hans (FP)	10 Spring St., Dexter	04930
Smith, Hugh A. (R)	Eastern Maine Med. Ctr., Bangor	04401
Striar, Ronald R. (PD)	94 Essex St., Bangor	04401
Strout, Warren G. (ANES)	83 Essex St., Bangor	04401
Taylor, H. Lewis (FP)	25 Church St., Dexter	04930
Thomas, Philip B. (ANES)	83 Essex St., Bangor	04401
Trowbridge, Mason, Jr. (IM)	142 Pine St., Bangor	04401
Tyson, Dudley B. (ANES)	91 Grove St., Bangor	04401
Vickers, Martyn A. (A,D)	268 State St., Bangor	04401
Vincze, Imre E. (FP)	St. Joseph Hosp., Bangor	04401
Vydas, Joseph (FP)	Bangor State Hosp., Bangor	04401
Wadsworth, Richard C. (PATH)	489 State St., Bangor	04401
Wagner, Samuel L. (FP)	2 Holmes St., Winterport	04496
Watt, Thomas L. (DE)	316 State St., Bangor	04401
Weisz, Hans (FP)	17 Sunrise Ter., Orono	04473
Wise, Joe R., Jr. (C)	1 Fern St., Bangor	04401
Wood, George W., III (IM,PUD)	156 No. Main St., Brewer	04412
Woodcock, John A. (ORS)	109 State St., Bangor	04401

HONORARY

Devan, Thomas A. (OO)	Palm Shores West, Apt. G-8, 830 North Shore Dr., St. Petersburg, Fla.	33701
Emerson, W. Merritt (FP,CD)	131 State St., Bangor	04401
Woodcock, Allan (OO)	109 State St., Bangor	04401

SENIOR

Adams, Asa C. (GS)	68 Main St., Orono	04473
Blaisdell, Carl E. (U)	336 Mt. Hope Ave., Bangor	04401
Butler, Harry (OTO)	77 Broadway, Bangor	04401

JUNIOR

Samuelson, Thomas W. (FP)	20 Eastfield Rd., Cape Elizabeth	04107

AFFILIATE

Dwyer, Clement S. (ANES)	35 Maple St., Bangor	04401

PISCATAQUIS COUNTY

President – Isaac Nelson, M.D.

Secretary-Treasurer – Robert C. Cornell, M.D.

ACTIVE

Bradbury, Francis W. (FP)	16 E. Main St., Dover-Foxcroft	04426
Cornell, Robert C. (ORS)	Box 518, Greenville	04441
Curtis, John B. (FP)	10 High St., Milo	04463
Garcia-Rey, Felix M. (FP)	Milo	04463
Howard, George C. (FP)	Oak St., Guilford	04443
Lightbody, Charles H. (FP)	No. Main St., Guilford	04443
Nelson, Isaac (FP)	Box 506, Greenville	04441
Neilson, Odd S. (AM,99)	120 Hudson Rd., Bangor	04401
Rodriguez, Araminta M. (FP)	Milo	04463
Stitham, Linus J. (FP,OBG)	50 Main St., Dover-Foxcroft	04426
Stone, Charles H., III (GS)	Box 498, Greenville	04441

HONORARY

Bundy, Harvey C. (OO)	42 Constitution Dr., Westbrook	04092
Nickerson, Norman H. (FP)	Greenville	04441
Stanhope, Charles N. (OO)	South St., Dover-Foxcroft	04426
Wyman, Edwin T. (OO)	Harvard Club of Boston, 374 Commonwealth Ave., Boston, Mass.	02215

SOMERSET COUNTY

President – Louis S. Hornstein, M.D.

Secretary-Treasurer – John H. Steeves, M.D.

ACTIVE

Amrein, H. Carl (FP,GS)	29 Weston Ave., Madison	04950
Briggs, Paul R. (GS)	Hartland	04943
Dow, John P. (FP)	Grove Hill, Pittsfield	04967
Hoch, Gretl J. (FP)	Phillips	04966
Hornstein, Louis S. (FP)	220 Water St., Skowhegan	04976
Jordan, W. Edward, Jr. (GS)	Box 218, Skowhegan	04976
Kaschub, Robert W. (IM)	R.F.D. No. 3 Skowhegan	04976
Kemezys, Kestutis M. (FP,ANES)	25 Garfield St., Madison	04950
Koopal, Soleiman (OBG)	Farview Ave., Skowhegan	04976
Laney, Richard P. (IM)	50 Water St., Skowhegan	04976
Reed, Howard L. (GS)	235 Madison Ave., Skowhegan	04976
Richards, Henry H. (FP)	32 Valley Rd., Cape Elizabeth	04107
Smith, Edgar J. (FP)	1 Park St., Fairfield	04937
Steeves, John H. (R)	Rt. 3 Skowhegan	04976
Stein, Ernest W. (FP)	72 Main St., Pittsfield	04967
Strickland, Marian L. (FP)	Easy St., Canaan	04924
Sullivan, George E. (ANES)	86 Summer St., Waterville	04901
Swett, Carlton E. (GS)	Box 526, Skowhegan	04976
Sy, Vincente L. (FP,U)	Milford Ave., Bingham	04920
Taylor, Richard C. (PATH)	Redington-Farview Gen. Hosp., Skowhegan	04976
Turner, Harland G. (FP,ANES)	Box 38, Norridgewock	04957

HONORARY

Lord, Maurice E. (OO)	Box 537, Lake Placid, Fla.	33852

SENIOR

Philbrick, Maurice S. (OO)	3349 N.W. 32nd Crt., Fort Lauderdale, Fla.	33309

WALDO COUNTY

President – John A. Caswell, M.D.

Secretary-Treasurer – Andrew J. Gay, M.D.

ACTIVE

Caswell, John A. (FP,GS)	16 Waldo Ave., Belfast	04915
Gay, Andrew J. (OPH)	Little River House, Belfast	04915
Hanbury, Euclid M. Jr., (GS)	Medical Bldg., Belfast	04915
Jollie, Peter M.	Fahey St., Belfast	04915
Knuuti, Harold E. (IM)	Medical Bldg., Belfast	04915
Lecher, Robert C. (FP)	3021 N. Hancock St., Colorado Springs, Col.	80907
Webber, John R. (FP)	6 Northport Ave., Belfast	04915

HONORARY

Small, Foster C. (FP)	169 High St., Belfast	04915
Stevens, Carl H. (FP)	18 Franklin St., Belfast	04915

SENIOR

Cobb, Norman E. (FP,GS)	Medical Bldg., Belfast	04915
Torrey, Raymond L. (FP)	R.F.D. No. 1 Belfast	04915

AFFILIATE

Temple, George L. (GS,ORS)	Fahey St., Belfast	04915

WASHINGTON COUNTY

President – George B. Shaw, M.D.

Secretary-Treasurer – Karl V. Larson, M.D.

ACTIVE

Bates, James C. (FP)	Eastport	04631
French, Rowland B. (FP,GS)	16 Water St., Eastport	04631
Jacob, Donald R. (FP)	Princeton	04668
Kazutow, John (GPM)	Box 113, Columbia Falls	04623
Larson, Karl V. (FP)	E. Machias	04630
MacBride, Robert G. (FP)	25 Washington St., Lubec	04652
Mason, Sabry E. (P.A.)-(FP)	Main St., Calais	04619
Mitchell, Hazen C. (FP,GS)	Calais	04619
Moore, Lawrence H. (FP)	Lubec Medical Ctr., Lubec	04652
Nackley, George N. (FP,GS)	1 School St., Machias	04654
Robertson, Donald M. (FP,GS)	Box 188, Milbridge	04658
Sears, Harold G. (FP,OM)	Second Ave., Woodland	04694
Shaw, George B. (FP)	27 Broadway, Machias	04654
Stott, Nelson W. (FP)	County Rd., Eastport	04631

HONORARY

Bennet, DaCosta F. (FP)	4 Main St., Lubec	04652
Mundie, Perley J. (OTO)	32 North St., Calais	04619

AFFILIATE

Kiel, Joseph B. (P)	Columbia Falls	04623

YORK COUNTY

President – Carl E. Richards, M.D.

Secretary-Treasurer – Charles W. Kinghorn, M.D.

Asst. Secretary-Treasurer – Melvin Bacon, M.D.

ACTIVE

Anton, Thomas (IM)	5 Graham St., Biddeford	04005
Bacon, Melvin (IM)	27 June St., Sanford	04073
Begenau, Vernon G. (ANES)	Post Rd., Wells	04090
Belmont, Ralph S. (FP)	6 Washington St., Sanford	04073
Berger, Steven (P)	Station B, Poughkeepsie, N.Y.	12602
Buell, William O. (OBG)	357 Elm St., Biddeford	04005
Charest, Leandre R. (GS)	314 Alfred St., Biddeford	04005
Cuneo, Kenneth J. (ANES)	31 Summer St., Kennebunk	04043
Derboven, Paul H. (FP)	120 Main St., Sanford	04073
Dorfman, Irvin (PATH)	Goodall Hosp., Sanford	04073
Dow, Owen O. (GS)	Box 388, Longwood Dr., Kennebunk	04043
Downing, J. Robert (FP)	11 Partridge Lane, Kennebunk	04043
Drummond, S. Dunton (FP)	Bar Mills	04004
Endicott, Ruth E. (FP)	Grasshopper Lane, Ogunquit	03907
Festino, Michael J. (IM)	66 Beach St., Saco	04072
Ficker, Robert F. (FP)	Maine St., Kennebunkport	04046
Fortier, Andre P. (FP,OBG)	68 Foss St., Biddeford	04005
Haas, Carl M. (OBG)	Box 546, Biddeford	04005
Haq, Badi M. (PATH)	Webber Hosp., Biddeford	04005
Hazzard, Lawrence R. (ANES)	Cider Hill Rd., York	03909
Hickey, Vincent J. (GS)	42 Bacon St., Biddeford	04005
Hill, Paul S., Jr. (FP,GS)	323 Main St., Saco	04072
Hoffman, Alvin A. (FP)	Box 38, York	03909
Hopkins, Herbert J. (FP,A)	24 Portland Ave., Old Orchard Beach	04064
Houle, Marcel P. (FP,GS)	200 Alfred St., Biddeford	04005
Johnston, James S. (FP,GS)	258 Main St., Saco	04072
LaFond, Robert S. (IM)	258 Main St., Saco	04072
Laltoo, Joseph M. (GS)	19 Hospital Dr., York	03909
Lapirow, Harry (IM)	99 Main St., Kennebunk	04043
Leigh, Kenneth E. (R)	Brixham Rd., York	03909
Lincourt, Armand S. (FP)	122 Main St., Sanford	04073
Lord, George A. (GS)	27 June St., Sanford	04073
Magaudda, Michael M. P. (FP,GS)	39 Old Orchard St., Old Orchard Beach	04064
Magocsi, Alexander W. (FP)	York	03909
Moore, Conner M. (PD)	372 Main St., Saco	04072
Moulton, Marion K. (FP)	W. Newfield	04095
Nieuwkerk, Willem F. (P)	Box 424, Kennebunkport	04046
O'Sullivan, William B. (FP,OBG)	Box 645, Biddeford	04005
Page, Lyman A. (PD)	372 Main St., Saco	04072
Palmer, Mahlon P. (OTO)	Nasson College, Springvale	04083
Patane, Joseph M. (FP,GS)	256 Alfred St., Biddeford	04005
Perry, Richard L. (R)	195 Main St., Sanford	04073
Peterlein, Walter R., Jr. (FP)	75 Main St., Springvale	04083
Rainforth, Douglas W. (GS)	27 June St., Sanford	04073

Richards, Carl E. (FP)	27 June St., Sanford	04073
Robert, Roger J. P. (ORS)	258 Main St., Saco	04072
Ross, Maurice (PD)	372 Main St., Saco	04072
Roussin, William T. (FP,CD)	48 Bacon St., Biddeford	04005
Scott, Arthur M., Jr. (IM,CD)	37 Amherst St., Biddeford	04005
Shaw, G. Patrick (OBG)	275 Main St., Biddeford	04005
Smith, Gerald R. (FP)	Ogunquit	03907
Smith, Oney P. (FP)	Post Road, Wells	04090
Taylor, Paul E. (FP)	9 Wentworth St., Kittery	03904
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Kinghorn, Charles W. (FP,OTO)	4 Wentworth St., Kittery	03904
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The figures in parentheses refer to County Societies as follows: (1) Androscoggin, (2) Aroostook, (3) Cumberland, (4) Franklin, (5) Hancock, (6) Kennebec, (7) Knox, (8) Lincoln-Sagadahoc, (9) Oxford, (10) Penobscot, (11) Piscataquis, (12) Somerset, (13) Waldo, (14) Washington, (15) York.

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Adams, Patricia Ann, Freeport 04032 (1)
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Pasternak, Irwin M., 230 French St., Bangor 04401 (10)
Patane, Joseph M., 256 Alfred St., Biddeford 04005 (15)
Patten, Roy S., 336 Mt. Hope Ave., Bangor 04401 (10)
Patterson, James, Apt. 10D, 45 Eastern Prom., Portland 04101 (3)
Paulding, Stephen B., 134 U.S. Rt. 1, Falmouth 04105 (3)
Pawle, Robert H., 251 U. S. Rt. 1, Falmouth 04105 (3)
Pearson, John J., 100 So. Main St., Old Town 04468 (10)
Pease, Horace B., Maine Coast Mem. Hosp., Ellsworth 04605 (5)
Peddie, Harry M. K., Doctors Park, 89 Hosp. St., Augusta 04330 (6)
Pendleton, Arthur D., 3 Green St., Fort Fairfield 04742 (2)
Pennoyer, Douglass C., 112 Vaughan St., Portland 04102 (3)
Penta, Walter E., 316 Woodford St., Portland 04103 (3)
Perry, Richard L., 195 Main St., Sanford 04073 (15)
Peterlein, Walter R., Jr., 75 Main St., Springvale 04083 (15)
Petterson, Herman C., Chebeague Island 04017 (3)
Pfeiffer, Paul H., Cherry Hill Ter., Waterville 04901 (6)
Phelps, Paulding, 13 Charles St., Portland 04102 (3)
Philbrick, Maurice S.,
3349 N. W. 32nd Crt., Fort Lauderdale, Fla. 33309 (12)
Phillips, David L., 191 Lincoln Ave., Rumford 04276 (9)
Phillips, Lewis E., 336 Mt. Hope Ave., Bangor 04401 (10)
Pines, Philip, Maine St., Limestone 04750 (2)
Plimpton, Jay R., 283 Water St., Augusta 04330 (6)
Pogue, Jackson S., 529 Gilmore Ave., Trafford, Pa. 15085 (3)
Poliner, Irving J., 95 West St., Portland 04102 (3)
Polisner, Saul R., 143 Vaughan St., Portland 04102 (3)
Pomerleau, Ovid F., 179 Main St., Waterville 04901 (6)
Pomerleau, Rodolphe J. F., Cascade Pk., Rt. 1, Waterville 04901 (6)
Porter, Edward C., 489 State St., Bangor 04401 (10)
Porter Joseph E., 22 Bramhall St., Portland 04102 (3)
Potts, Ronald S., Central Maine Gen. Hosp., Lewiston 04240 (1)
Poulin, Albert A., Cherry Hill Dr., Waterville 04901 (6)
Poulin, James E., 177 Main St., Waterville 04901 (6)
Powell, Ralph C., Damariscotta 04543 (8)
Pratt, Loring W., 325 Kennedy Dr., Waterville 04901 (6)
Price, Richard D., R.F.D. 2, Caribou 04736 (2)
Proctor, Ray A., Garden Circle, Caribou 04736 (2)
Proctor, Thomas E., Boothbay Harbor 04538 (8)
Proudian, Paul O., 776 Main St., Westbrook 04092 (3)
Proulx, Harvey J., 185 Webster St., Lewiston 04240 (1)
Purinton, William A., St. Joseph Hosp., Bangor 04401 (10)

Rainforth, Douglas W., 27 June St., Sanford 04073 (15)
Rand, Carleton H., 219 Oak St., Lewiston 04240 (1)
Rando, Joseph J., 111 Webster St., Lewiston 04240 (1)
Ray, Ferris S., 7 Bramhall St., Portland 04102 (3)
Read, Frank W., 9 Deering St., Portland 04101 (3)
Reed, David G., 7 Washington St., Camden 04843 (7)
Reed, Howard L., 235 Madison Ave., Skowhegan 04976 (12)
Reed, James W., 18 Main St., Farmington 04938 (4)
Reel, John J., 59 So. Front St., Richmond 04357 (6)
Reeves, Edward L., 179 Sabattus St., Lewiston 04240 (1)
Reeves, Helene M., 100 Locksley Rd., Auburn 04210 (1)
Rerrick, Erwin G., Box C, Pownal 04069 (3)
Reynolds, Arthur P., 29 Second St., Presque Isle 04769 (2)
Reynolds, John F., 325 Kennedy Dr., Waterville 04901 (6)
Reynolds, Ralph L., 325 Kennedy Mem. Dr., Waterville (6)
Rice, John D., Jr., 144 State St., Portland 04101 (3)
Richards, A. Dewey, 11 Gage St., Bridgton 04009 (3)
Richards, Carl E., 27 June St., Sanford 04073 (15)
Richards, Henry H., 32 Valley Rd., Cape Elizabeth 04107 (12)
Richards, Lee W., Jr., 89 Hospital St., Augusta 04330 (6)
Rideout, Samuel, Green St., Fort Fairfield 04742 (2)
Ridlon, Joseph R., 58 South St., Gorham 04038 (3)
Robert, Roger J. P. 258 Main St., Saco 04072 (15)
Robertson, Donald M., Box 188, Milbridge 04658 (14)
Robertson, George J., 1370 Turnpike St., North Andover, Mass. 01845 (6)
Robinson, Hugh P., 229 Vaughan St., Portland 04102 (3)
Rock, Daniel A., 477 Main St., Lewiston 04240 (1)
Rodriguez, Araminta M., Milo 04463 (11)
Rodriguez, Jose M., 325 Kennedy Dr., Waterville 04901 (6)
Rogers, Albert M., 48 Gilman St., Portland 04102 (3)
Rohm, Walter, Augusta State Hosp., Augusta 04330 (6)
Root, John A., 22 White St., Rockland 04841 (7)
Rosenblatt, Stanley D., 480 Main St., Lewiston 04240 (1)
Ross, Maurice, 372 Main St., Saco 04072 (15)
Roussin, William T., 48 Bacon St., Biddeford 04005 (15)
Rowan, Gilbert R., 10 Oak Grove Ave., Bath 04530 (8)
Rowe, Linwood M., Rumford Com. Hosp., Rumford 04276 (9)
Royal, Albert P., Jr., 82 Maine Ave., Rumford 04276 (9)
Rubins, Nina B., E. A. Center Mem. Clinic, Steep Falls 04085 (3)
Rubins, Talivaldis, E. A. Center Mem. Clinic, Steep Falls 04085 (3)
Russell, Daniel F. D., Leeds 04263 (1)
Russell, Robert F., Castine 04421 (5)

Russell, Theodore M., Doctors Park, 89 Hospital St., Augusta 04330 (6)
Ryan, Patrick J.M., 345 Water St., Gardiner 04345 (6)
Rynne, Michael V., 191 Lincoln Ave., Rumford 04276 (9)

Sager, George F., 18 Bramhall St., Portland 04102 (3)
Samuelsen, Thomas W., 20 Eastfield Rd., Cape Elizabeth 04107 (10)
Sandvoss, Herman G., Union St., Kennebunkport 04046 (15)
Sanfacon, Philip G., 407B Dalton Dr. Winooski, Vt. 05404 (2)
Sanford, Theodore H., (P.A.), St. Mary's Medical Office Bldg.
97 Campus Ave., Lewiston 04240 (1)
Santoro, Domenico A., 43 Deering St., Portland 04101 (3)
Sanzenbacher, Karl E., 325C Kennedy Mem. Dr., Waterville 04901 (6)
Satir, Ahmet, Box 682, Augusta 04330 (6)
Saunders, Allen I., Ferry Rd., R.F.D. 2, Augusta 04330 (6)
Saunders, Norman W., 233 Vaughan St., Portland 04102 (3)
Saunders, Sallie H., R.F.D., Camden 04843 (7)
Sawyer, Howard P., Jr., 22 Bramhall St., Portland 04102 (3)
Sbaschnig, Robert J., Central Maine Gen. Hosp., Lewiston 04240 (1)
Schall, David W., 56 Baribeau Dr., Brunswick 04011 (8)
Schmidt, Lorrimer M., 13 Elm St., Augusta 04330 (6)
Schnittke, Sidney M., Porter Ave., W., Rumford 04276 (9)
Schroder, John C., 205 French St., Bangor 04401 (10)
Schumacher, William E., 14 Westwood Rd., MD "B", Augusta 04330 (6)
Schwarz, Harald J., Seton Hospital, Waterville 04901 (6)
Scolten, Adrian H., 32 Deering St., Portland 04101 (3)
Scott, Arthur M., Jr., 37 Amherst St., Biddeford 04005 (15)
Sears, Harold G., Second Ave., Woodland 04694 (14)
Seligman, Morris J., Veterans Adm., Togus 04330 (6)
Selmeci, Tibor G., Hayden Ct., Woodstock, N. B., Can. (2)
Selvage, Irving L., Jr., 22 Bramhall St., Portland 04102 (3)
Senenky, Joseph P., Augusta State Hosp., Augusta 04330 (6)
Sensenig, David M., 431 State St., Bangor 04401 (10)
Serrage, John C., 229 Vaughan St., Portland 04102 (3)
Sewall, Elmer M., 14 Park St., Orono 04473 (10)
Sewall, Kenneth W., 2 School St., Waterville 04901 (6)
Shapero, Benjamin L., 431 State St., Bangor 04401 (10)
Shapiro, Morrill, 7 Bramhall St., Portland 04102 (3)
Shaw, G. Patrick, 275 Main St., Biddeford 04005 (15)
Shaw, George B., 27 Broadway, Machias 04654 (14)
Shaw, John H., 131 Sewall St., Augusta 04330 (6)
Sheehan, Terrance J., Doctors Park, 89 Hospital St., Augusta 04330 (6)
Shelton, M. Tieche, 21 Western Ave., Augusta 04330 (6)
Shelton, Robert L., 21 Western Ave., Augusta 04330 (6)
Shems, Albert, 313 Main St., Lewiston 04240 (1)
Sherman, Fuller G., Spruce Pt., Boothbay Harbor 04538 (8)
Shields, Daniel R., 369 Main St., Lewiston 04240 (1)
Shields, Thomas F., 300 Pine St., Lewiston 04240 (1)
Shubert, Alice J., 125 Leighton St., Bangor 04401 (10)
Shubert, William M., 336 Mt. Hope Ave., Bangor 04401 (10)
Shurman, Hans, 10 Spring St., Dexter 04930 (10)
Sidwell-Thompson, Doris M.,
R.F.D. Whittier Rd., W. Ossipee, N. H. 03890 (3)
Sieling, Walter H., Jr., Upper Round Pond Rd., Bristol 04539 (8)
Sigafoos, J. Harvey, Pleasant Point 04563 (7)
Silver, Randall H., Maine Coast Mem. Hosp., Ellsworth 04605 (5)
Simon, Pedro T., 373 Main St., Caribou 04736 (2)
Simpson, Margaret R., 2 Sea Barn Rd., Cape Elizabeth 04107 (6)
Skillin, Charles E., 111 Westcott Rd., South Portland 04106 (3)
Sleeper, Francis H., 3 Colony Rd., Augusta 04330 (6)
Small, Foster C., 169 High St., Belfast 04915 (13)
Smith, Carroll H., Box 785, Presque Isle 04769 (2)
Smith, Christopher S., Box 232, Farmington 04938 (4)
Smith, Edgar J., 1 Park St., Fairfield 04937 (12)
Smith, Gerald R., Ogunquit 03907 (15)
Smith, Hugh A., Eastern Maine Med. Ctr., Bangor 04401 (10)
Smith, Jacob, 709 High St., Bath 04530 (8)
Smith, James O., 118 Front St., Bath 04530 (8)
Smith, Kenneth E., Veterans Adm., Togus 04330 (6)
Smith, Marshall E., Pratt Rd., Caribou 04736 (2)
Smith, Oney P., Post Rd., Wells 04090 (15)
Sokol, Stephen A., Central Maine Gen. Hosp., Lewiston 04240 (1)
Somerville, Robert B., 45 Hillside St., Presque Isle 04769 (2)
Sommer, Robert G., 7 Bramhall St., Portland 04102 (3)
Soreff, Stephen M., Maine Medical Ctr., Portland 04102 (3)
Southall, Rogers C., 157 Pine St., Portland 04102 (3)
Sowles, Horace K., 413 Blackstrap Rd., Falmouth 04105 (3)
Spear, William, R.F.D. No. 2, Sabattus 04280 (1)
Spellman, Francis A., Veterans Adm., Togus 04330 (6)
Stanhope, Charles N., South St., Dover-Foxcroft 04426 (11)
Starks, Pauline G., Pemaquid Point 04561 (8)
Steele, Charles W., 472 Main St., Lewiston 04240 (1)
Steeves, John H., Rt. 3, Skowhegan 04976 (12)
Stein, Ernest W., 72 Main St., Pittsfield 04967 (12)
Stephenson, Richard B., Bldg. 1,
Rm. 118, National Institutes of Health, Bethesda, Md. 20014 (3)
Stetkevych, Alexander G., 1 Meadow Rd., Chatham, N. J. 07928 (8)
Stevens, Carl H., 18 Franklin St., Belfast 04915 (13)
Stevens, Harold W., 369 Ferry Rd., Saco 04072 (15)
Stevens, Theodore M., 148 State St., Portland 04101 (3)
Stewart, Nancy H., Hancock St., Bar Harbor 04609 (5)
Stewart, Richard D., 1 North St., Bath 04530 (8)
Stewart, Winston G., Hancock St., Bar Harbor 04609 (5)
Stimson, Barbara B., Star Route 22-282, Owl's Head 04854 (7)
Stinchfield, Allan J., Box 343, Augusta 04330 (6)
Stitham, Linus J., 50 Main St., Dover-Foxcroft 04426 (11)
Stocks, Joseph F., 22 Bramhall St., Portland 04102 (3)
Stone, Charles H., III, Box 498, Greenville 04441 (11)
Storer, Daniel P., 108 Fessenden St., Portland 04103 (3)
Stott, Nelson W., County Rd., Eastport 04631 (14)
Strach, Toffield B. J., 3 Deering St., Portland 04101 (3)
Strauss, William T., Box 547, Brunswick 04011 (3)
Striar, Ronald R., 94 Essex St., Bangor 04401 (10)
Strickland, Marian L., Easy St., Canaan 04924 (12)
Stroud, Geoffrey A., 65 Baribeau Dr., Brunswick 04011 (3)
Strout, Warren G., 83 Essex St., Bangor 04401 (10)
Stubing, Peter R., 106 Campus Ave., Lewiston 04240 (1)
Stucki, Paul, 325 Kennedy Dr., Waterville 04901 (6)
Sturtevant, Vaughn R., 325 Kennedy Dr., Waterville 04901 (6)
Sube, Janis, 108 Elm St., Camden 04843 (7)
Sullivan, George E., 86 Summer St., Waterville 04901 (12)
Sundaram, Venkat R., 87A Fish St., Turner 04282 (1)
Suyama, Eji, 58 W. Main St., Ellsworth 04605 (5)
Swanson, Ronald A., Regional Mem. Hosp., Brunswick 04011 (8)
Sweatt, Linwood A., 48 Drummond St., Auburn 04210 (1)
Swengel, Richard M., 477 Main St., Lewiston 04240 (1)
Swett, Alfred E., 20 Parsons Rd., Portland 04103 (3)
Swett, Carlton E., Box 526, Skowhegan 04976 (12)
Swett, Clyde I., 18 Sherman St., Island Falls 04747 (2)
Sy, Vincente L., Milford Ave., Bingham 04920 (12)
Sylvester, Stanley B., Box 548, Portland 04112 (3)
Szelenyi, Ernest, Box C, Pownal 04069 (3)

Tabachnick, Henry M., 110 Park Ave., Portland 04101 (3)
Tai, Tse-Wu, Rumford Comm. Hosp., Rumford 04276 (9)
Takach, Robert J., 325A Kennedy Dr., Waterville 04901 (6)
Tao, Zui S., Main St., Fort Kent 04743 (2)
Tardif, Lionel R., 9 Campus Ave., Lewiston 04240 (1)
Taxiarchis, Louis N., R.F.D. No. 1, West Buxton 04093 (3)
Taylor, H. Lewis, 25 Church St., Dexter 04930 (10)
Taylor, Paul E., 9 Wentworth St., Kittery 03904 (15)
Taylor, Richard C., Redington-Fairview Gen. Hosp., Skowhegan 04976 (12)
Taylor, Richard W., St. Mary's Gen. Hosp., Lewiston 04240 (1)
Taylor, William C., Pineland Hosp. & Training Ctr., Pownal 04069 (3)
Taylor, William F., 134 U.S. Route 1, Falmouth 04105 (3)
Tchao, Jou S., 181 Russell St., Lewiston 04240 (1)
Telfeian, Alphonse, 321 Brackett St., Portland 04102 (3)
Temple, George L., Fahey St., Belfast 04915 (13)
Tetreau, William J., 111 Westcott Rd., South Portland 04106 (3)
Thacher, Henry C., 117 Goff St., Auburn 04210 (1)
Thegen, W. Edward, Elm St., Bucksport 04416 (5)
Thomas, Philip B., 83 Essex St., Bangor 04401 (10)
Thompson, Philip P., Jr., 131 Chadwick St., Portland 04102 (3)
Thorpe, Darrell P., Maine Medical Ctr., Portland 04102 (3)
Tibbetts, Otis B., 181 Gamage Ave., Auburn 04210 (1)
Tibbetts, Otis P., Central Maine Gen. Hosp., Lewiston 04240 (1)
Timothy, Robert P., 229 Vaughan St., Portland 04102 (3)
Tiongson, Antonio C., 29 Malo St., Lewiston 04240 (1)
Tiongson, Cornelia M., 185 Webster St., Lewiston 04240 (1)
Titherington, John B., Shore Rd., Bremen 04551 (3)
Torrey, Raymond L., R.F.D. No. 1, Belfast 04915 (13)
Tounge, Harry G., Jr., 12 Union St., Camden 04843 (7)
Tousignant, Camille, 111 Pine St., Lewiston 04240 (1)
Toussaint, Leonid G., Box 9, Fort Kent 04743 (2)
Towne, Charles E., 18 Common St., Waterville 04901 (6)
Towne, John W., 325C Kennedy Mem. Dr., Waterville 04901 (6)
Tracy, Mary J., Bristol Rd., Damariscotta 04543 (8)
Trask, Henry M., 24 Hersey St., Portland 04103 (3)
Trembly, Bruce, 325 Kennedy Dr., Waterville 04901 (6)
Trowbridge, Mason, Jr., 142 Pine St., Bangor 04401 (10)
Tsao, Wu-Ming, Veterans Adm., Togus 04330 (6)
Turcotte, Guy N., 7 Bramhall St., Portland 04102 (3)
Turcotte, Richard W., 95 Campus Ave., Lewiston 04240 (1)
Turgeon, Raphael F., 367 Main St., Westbrook 04092 (3)
Turnbull, Elliott D., Elm House, Naples 04055 (3)
Turner, Fennell P., Veterans Adm. Ctr., Togus 04330 (6)
Turner, Harland G., Box 38, Norridgewock 04957 (12)
Turville, Charles S., Box E, Alfred 04002 (15)
Twadelle, Frank W., 345 Water St., Gardiner 04345 (6)
Tyler, J. Wayne, 222 Pine St., Lewiston 04240 (1)
Tyson, Dudley B., 91 Grove St., Bangor 04401 (10)

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Urjanis, Janis, 710 Cannons Lane, Louisville, Ky. 40206 (3)

Vachon, Robert D., 34 Winter St., Sanford 04073 (15)
Van Deventer, Wilhelm H. J.,
R.F.D. 1, Mere Point Rd., Brunswick 04011 (3)
Van Lonkhuyzen, Maurice, 131 State St., Portland 04101 (3)
Van Pelt, John C., 50 Union St., Ellsworth 04605 (5)
Veilleux, Lucien F., 325 Kennedy Dr., Waterville 04901 (6)
Vickers, Martyn A., 268 State St., Bangor 04401 (10)
Viger, Leopold A., 10 Amherst St., Biddeford 04005 (15)
Viles, Wallace E., Turner 04282 (1)
Villandry, Philip J., 22 Bramhall St., Portland 04102 (3)
Vincze, Imre E., St. Joseph Hosp., Bangor 04401 (10)
Voss, Carlyle B., 22 Bramhall St., Portland 04102 (3)
Vydas, Joseph, Bangor State Hosp., Bangor 04401 (10)

Wadsworth, Richard C., 489 State St., Bangor 04401 (10)
Wakana, Minoru, 33 Lyndon St., Caribou 04736 (2)
Walker, Douglass W., Maine Medical Ctr., Portland 04102 (3)
Wagner, Samuel L., 2 Holmes St., Winterport 04496 (10),
Wakefield, Robert D., St. Mary's Hosp., Lewiston 04240 (1)
Walsh, Andrew C., 144 State St., Portland 04101 (3)
Ward, John V., 8 Waites Landing Rd., Falmouth Foreside 04105 (3)
Ward, William W., Box 646, Rockland 04841 (7)
Ware, Roland G., Jr., 22 Bramhall St., Portland 04102 (3)
Warren, Henry S., Derby Rd., Islesboro 04848 (7)
Wasgatt, Wesley N., 41 Talbot Ave., Rockland 04841 (7)
Watanabe, Tatsuo, 325 Kennedy Mem. Dr., Waterville 04901 (6)
Waterman, Dorothy, Waldoboro 04572 (7)
Waterman, Richard, Waldoboro 04572 (7)
Watt, Thomas L., 316 State St., Bangor 04401 (10)
Weaver, Michael L., 10 Water St., Brunswick 04011 (3)
Webber, Isaac M., 29 Deering St., Portland 04101 (3)
Webber, John R., 6 Northport Ave., Belfast 04915 (13)
Webber, Peter B., 233 Vaughan St., Portland 04102 (3)
Webber, Wedgwood P., 460 Main St., Lewiston 04240 (1)
Weisz, Hans, 17 Sunrise Ter., Orono 04473 (10)
Wheelwright, Henry J., Augusta Gen. Hosp., Augusta 04330 (6)
White, Chester W., Jr., 22 Bramhall St., Portland 04102 (3)
White, Henry O., 22 White St., Rockland 04841 (7)
White, Leland M., 18 Pleasant St., Caribou 04736 (2)
White, Richard L., 7 Bramhall St., Portland 04102 (3)
White, William J., 1 Mitchell Rd., South Portland 04106 (3)
Whitney, Philip G., 233 Vaughan St., Portland 04102 (3)
Whittier, Alice A. S., 143 Neal St., Portland 04102 (3)
Wieting, William F., 390 State St., Portsmouth, N.H. 03801 (15)
Wight, Donald G., 30 Mitchell Rd., South Portland 04106 (3)
Wilbur, Herbert T., Jr., 100 Main St., Southwest Harbor 04679 (5)
Wilder, William D., Box 2146, Augusta 04330 (6)
Williams, Edward P., 72 Main St., Houlton 04730 (2)
Williams, James A., 39 Pleasant St., Mechanic Falls 04256 (1)
Williams, Thomas W., 22 White St., Rockland 04841 (7)
Williamson, Elizabeth E., Blue Hill 04614 (5)
Williamson, Russell G., Blue Hill Mem. Hosp., Blue Hill 04614 (5)
Wilson, G. Ivan, 48 Court St., Houlton 04730 (2)
Wilson, Robert D., Mt. Desert Island Hosp., Bar Harbor 04609 (5)
Wilson, Robert W., Veterans Adm., Togus 04330 (6)
Winchenbach, Francis A., 910 Washington St., Bath 04530 (8)
Winkelbauer, Rudolf G., Baribeau Dr., Brunswick 04011 (3)
Wise, Joe R., Jr., 1 Fern St., Bangor 04401 (10)
Wood, George W., III, 156 North Main St., Brewer 04412 (10)
Woodcock, Allan, 109 State St., Bangor 04401 (10)
Woodcock, John A., 109 State St., Bangor 04401 (10)
Woodruff, Alan F., 16 Summer St., Rockland 04841 (7)
Woodman, Arthur B., 390 Copperfield Rd., Spring Hill, Fla. 33512 (3)
Worthing, Verla E., Box A., Thomaston 04861 (7)
Wren, James C., Veterans Adm. Ctr., Togus 04330 (6)
Wright, Herbert J. Jr., 45 Golder St., Lewiston 04240 (1)
Wyman, David S., 233 Vaughan St., Portland 04102 (3)
Wyman, Edwin T., Harvard Club of Boston, 374 Commonwealth Ave.,
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Yaghmai, Madjid, Cary Mem. Hosp., Caribou 04736 (2)
York, Elihu, 65 Baribeau Dr., Brunswick 04011 (8)
Young, E. Stanley, Poland Spring 04274 (1)
Young, John, Paradise Rd., Bethel 04217 (9)
Young, William J., Mercy Hosp., Portland 04101 (3)

Zanca, Ralph, 185 Webster St., Lewiston 04240 (1)
Zeller, Alan W., Main St., Newcastle 04553 (8)
Zerner, John, 49 Deering St., Portland 04101 (3)
Zolov, Benjamin, 296 Congress St., Portland 04101 (3)

PAST PRESIDENTS

Maine Medical Association

*Isaac Lincoln, M.D., Brunswick	April-June, 1853
*James McKeen, M.D., Topsham	1853-1854
*Charles Millett, M.D., Lewiston	1854-1855
*Joseph H. Estabrook, M.D., Camden	1855-1856
*Hosea Rich, M.D., Bangor	1856-1857
*Gilman Daveis, M.D., Portland	1857-1858
*J. C. Bradbury, M.D., Old Town	1858-1859
*H. H. Hill, M.D., Augusta	1859-1860
*T. G. Stockbridge, M.D., Bath	1860-1861
*H. M. Harlow, M.D., Augusta	1861-1862
*Alonzo Garcelon, M.D., Lewiston	1862-1863
*J. T. Gilman, M.D., Portland	1863-1864
*N. P. Monroe, M.D., Belfast	1864-1865
*Amos Nourse, M.D., Bath	1865-1866
*S. H. Tewksbury, M.D., Portland	1866-1867
*Cyrus Briggs, M.D., Augusta	1867-1868
*I. T. Dana, M.D., Portland	1868-1869
*D. McRuer, M.D., Bangor	1869-1870
*B. F. Buxton, M.D., Warren	1870-1871
*A. J. Fuller, M.D., Bath	1871-1872
*A. P. Snow, M.D., Winthrop	1872-1873
*A. F. Page, M.D., Bucksport	1873-1874
*Thomas H. Brown, M.D., Paris	1874-1875
*J. H. Bates, M.D., Yarmouth	1875-1876
*E. F. Sanger, M.D., Bangor	1876-1877
*T. H. Jewett, M.D., South Berwick	1877-1878
*M. C. Wedgwood, M.D., Lewiston	1878-1879
*S. C. Gordon, M.D., Portland	1879-1880
*William Warren Greene, M.D., Portland	1880-1881
*A. K. P. Meserve, M.D., Buxton	1881-1882
*George E. Brickett, M.D., Augusta	1882-1883
*Oren A. Horr, M.D., Lewiston	1883-1884
*Thomas A. Foster, M.D., Portland	1884-1885
*Sumner Laughton, M.D., Bangor	1885-1886
*J. B. Walker, M.D., Thomaston	1886-1887
*Frederick C. Thayer, M.D., Waterville	1887-1888
*Stephen H. Weeks, M.D., Portland	1888-1889
*Benjamin F. Sturgis, M.D., Auburn	1889-1890
*Samuel B. Hunter, M.D., Machias	1890-1891
*Edwin M. Fuller, M.D., Bath	1891-1892
*Alfred Mitchell, M.D., Brunswick	1892-1893
*John A. Donovan, M.D., Lewiston	1893-1894
*W. P. Giddings, M.D., Gardiner	1894-1895
*Lewis W. Pendleton, M.D., Portland	1895-1896
*D. A. Robinson, M.D., Bangor	1896-1897
*Wallace K. Oakes, M.D., Auburn	1897-1898
*Charles O. Hunt, M.D., Portland	1898-1899
*Bigelow T. Sanborn, M.D., Augusta	1899-1900
*Edward H. Hill, M.D., Lewiston	1900-1901
*Frederic H. Gerrish, M.D., Portland	1901-1902
*Hiram Hunt, M.D., Greenville	1902-1903
*Augustus S. Thayer, M.D., Portland	1903-1904
*F. L. Dixon, M.D., Lewiston	1904-1905
*Randall D. Bibber, M.D., Bath	1905-1906
*C. E. Williams, M.D., Auburn	1906-1907
*B. B. Foster, M.D., Portland	1907-1908
*Alfred D. Sawyer, M.D., Fort Fairfield	1908-1909
*Galen M. Woodcock, M.D., Bangor	1909-1910
*E. H. Bennett, M.D., Lubec	1910-1911
*Stanley P. Warren, M.D., Portland	1911-1912
*Ralph H. Marsh, M.D., Guilford	1912-1913
*W. C. Peters, M.D., Bangor	1913-1914
*H. L. Bartlett, M.D., Norway	1914-1915
*Erastus E. Holt, M.D., Portland	1915-1916
*W. F. Hart, M.D., Camden	1916-1917
*James A. Spalding, M.D., Portland	1917-1918
*George H. Coombs, M.D., Waldoboro	1918-1919
*H. B. Mason, M. D., Calais	1919-1920
*Theodore E. Hardy, M.D., Waterville	1920-1921
*Addison S. Thayer, M.D., Portland	1921-1922
*L. T. Snipe, M.D., Bath	1922-1923
*C. A. Moulton, M.D., Hartland	1923-1924
*F. W. Mann, M.D., Houlton	1924-1925
*J. D. Phillips, M.D., Southwest Harbor	1925-1926
*L. P. Gerrish, M.D., Lisbon Falls	1926-1927
*Herbert F. Twitchell, M.D., Portland	1927-1928
*Frank Y. Gilbert, M.D., Portland	1928-1929
*Delbert M. Stewart, M.D., South Paris	1929-1930
*Charles B. Sylvester, M.D., Portland	1930-1931
*Ernest V. Call, M.D., Lewiston	1931-1932
*E. Delmont Merrill, M.D., Dover-Foxcroft	1932-1933
*Warren E. Kershner, M.D., Bath	1933-1934
*Edwin W. Gehring, M.D., Portland	1934-1935
*John L. Johnson, M.D., Bangor	1935-1936
*Frederick T. Hill, M.D., Waterville	1936-1937
*Ralph W. Wakefield, M.D., Bar Harbor	1937-1938
Willard H. Bunker, M.D., York Harbor	1938-1939
*George L. Pratt, M.D., Fairfield	1939-1940
*Thomas A. Foster, M.D., Portland	1940-1941
*P. L. B. Ebbett, M.D., Houlton	1941-1942
Carl H. Stevens, M.D., Belfast	1942-1943
*Oscar F. Larson, M.D., Machias	1943-1944
*R. V. N. Bliss, M.D., Blue Hill	1944-1945
*Adam P. Leighton, M.D., Portland	1945-1946
*John O. Piper, M.D., Waterville	1946-1947
*Stephen A. Cobb, M.D., Sanford	1947-1948
*Forrest B. Ames, M.D., Bangor	1948-1949
Ralph A. Goodwin, Sr., M.D., Auburn	1949-1950
Foster C. Small, M.D., Belfast	1950-1951
*C. Harold Jameson, M.D., Rockland	1951-1952
*Eugene H. Drake, M.D., Portland	1952-1953
Norman H. Nickerson, M.D., Greenville	1953-1954
*Robert W. Belknap, M.D., Damariscotta	June-August 1954(Died in Office)
*William F. Mahaney, M.D., Saco	1954-1955
Martyn A. Vickers, M.D., Bangor	1955-1956
*Armand Albert, M.D., Van Buren	1956-1957
Francis A. Winchenbach, M.D., Bath	1957-1958
Eugene E. O'Donnell, M.D., Portland	1958-1959
Allan Woodcock, M.D., Bangor	1959-1960
*Wilson H. McWethy, M.D., Augusta	June 1960-February 1961 (Died in Office)
Carl E. Richards, M.D., Sanford	February 1961-June 1961
James A. MacDougall, M.D., Rumford	1961-1962
*Ralph C. Stuart, M.D., Guilford	1962-1963
Ernest W. Stein, M.D., Pittsfield	1963-1964
Thomas A. Martin, M.D., Portland	1964-1965
John F. Dougherty, M.D., Bath	1965-1966
George E. Sullivan, M.D., Fairfield	1966-1967
Paul S. Hill, Jr., M.D., Saco	1967-1968
Asa C. Adams, M.D., Orono	1968-1969
Charles F. Branch, M.D., Auburn	1969-1970
Charles R. Glassmire, M.D., Portland	1970-1971
Linus J. Stitham, M.D., Dover-Foxcroft	1971-1972

*Deceased

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Safety: An excellent clinical record. In general use, the most common side effects reported have been drowsiness, ataxia and confusion, particularly in the elderly and debilitated.

in relief of clinically significant anxiety

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(chlordiazepoxide HCl)

5-mg, 10-mg, 25-mg capsules
up to 100 mg daily in severe anxiety

Before prescribing, please consult complete product information, a summary of which follows:
Indications: Relief of anxiety and tension occurring alone or accompanying various disease states.
Contraindications: Patients with known hypersensitivity to the drug.
Warnings: Caution patients about possible combined effects with alcohol and other CNS depressants. As with all CNS-acting drugs, caution patients against hazardous occupations requiring complete mental alertness (*e.g.*, operating machinery, driving). Though physical and psychological dependence have rarely been reported on recommended doses, use caution in administering to addiction-prone individuals or those who might increase dosage; withdrawal symptoms (including convulsions), following discontinuation of the drug and similar to those seen with barbiturates, have been reported. Use of any drug in pregnancy, lactation, or in women of childbearing age requires that its potential benefits be weighed against its possible hazards.
Precautions: In the elderly and debilitated, and in children over six, limit to smallest effective dosage (initially 10 mg or less per day) to preclude ataxia or oversedation, increasing gradually as needed and tolerated. Not recommended in children under six. Though generally not recommended, if combination therapy with other psychotropics seems indicated, carefully consider individual pharmacologic effects, particularly in use of potentiating drugs such as MAO inhibitors and phenothiazines. Observe usual precautions in presence of impaired renal or hepatic function. Paradoxical reactions (*e.g.*, excitement, stimulation and acute rage) have been reported in psychiatric patients and hyperactive aggressive children. Employ usual precautions in treatment of anxiety states with evidence of impending depression; suicidal tendencies may be present and protective measures necessary. Variable effects on blood coagulation have been reported very rarely in patients receiving the drug and oral anticoagulants; causal relationship has not been established clinically.
Adverse Reactions: Drowsiness, ataxia and confusion may occur, especially in the elderly and debilitated. These are reversible in most instances by proper dosage adjustment, but are also occasionally observed at the lower dosage ranges. In a few instances syncope has been reported. Also encountered are isolated instances of skin eruptions, edema, minor menstrual irregularities, nausea and constipation, extrapyramidal symptoms, increased and decreased libido—all infrequent and generally controlled with dosage reduction; changes in EEG patterns (low-voltage fast activity) may appear during and after treatment; blood dyscrasias (including agranulocytosis), jaundice and hepatic dysfunction have been reported occasionally, making periodic blood counts and liver function tests advisable during protracted therapy.
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